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EQUINE ENCEPHALOMYELITIS IN MASSACHUSETTS*

An Analysis of the 1938 Outbreak, a Follow-Up of Cases and a Report of a Mosquito Survey

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BOSTON

INFECTION of man with the Eastern variety of equine encephalomyelitis was first demonstrated by Fothergill, Dingle, Farber and Connerley[1] and by Webster and Wright.[2] These workers isolated the virus from the brains of children who had died of a previously unrecognized type of encephalitis during an outbreak of equine encephalomyelitis in the horses of southeastern Massachusetts. Reviews of equine-encephalomyelitis infections in horses and men and the history of this disease have been made by Gordon[3] and Fothergill.[4] A description of the Massachusetts outbreak of equine encephalomyelitis, with an analysis of the cases in man, was reported by Feemster[5] in 1938. It is the purpose of this paper to add further detail to Feemster's report, to follow up the surviving patients, and to make a report on the Massachusetts Mosquito Survey, which was organized to ascertain the distribution of mosquitoes that have been demonstrated to transmit experimentally the Eastern type of equine encephalomyelitis to laboratory animals.

THE MASSACHUSETTS OUTBREAK

In July, 1938, for the first time, equine encephalomyelitis was recognized in horses in southeastern Massachusetts; and in the course of about ten weeks 269 deaths were reported. The geographical distribution of deaths in horses is given in Figure 1. The disease made its first appearance in the upper basin of the Taunton River, which drains through nearby Rhode Island, and spread to contiguous areas, particularly toward the northeast. There were relatively few cases to the south and southeast, and none occurred on Cape Cod or the nearby islands. About 70 per cent of the Massachusetts cases occurred in a thirty-mile square, extending from the mouth of the Taunton River in the south to Boston on the north, and from the boundary of Rhode Island, eastward to the coast of Massachusetts. Isolated cases occurred as far north as Maine and as far west as Worcester. Rhode Island to the west reported 55 cases among horses, and Connecticut 29.

During the summer and early autumn, rainfall in southeastern Massachusetts and Rhode Island was unusually heavy, and as a result mosquitoes were very prevalent in these areas. The prevailing winds in this region are from the southwest, and the tendency of the outbreak to spread in a northeasterly direction may be partly explained by the supposition that infection-bearing mosquitoes were carried by these prevailing winds. Two other factors, the movement of infected horses and the flights of infected birds, probably played an important role in the spread of the outbreak. Horses that were apparently well were moved from one country fair to another; on one such occasion a horse was moved thirty miles northeast to a town near the coast. Although apparently well when moved, the horse developed symptoms on the following day, and the diagnosis of equine encephalomyelitis was proved by the isolation of the virus from the brain. During the outbreak, Fothergill and Dingle[6] isolated the Eastern type of virus from the brains of a pigeon that had died in the same area, southeastern Massachusetts, and Tyzzer, Sellards and Bennett[7] isolated the same virus from the brains of ring-necked pheasants from Connecticut. These findings suggested that birds may be reservoirs of the disease and factors in its spread.

Almost simultaneously, in the same area, a new type of human encephalitis appeared, largely limited to children. The geographical distribution of the cases is shown in Figure 2. This outbreak was the first known infection of man with the

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Eastern variety of equine encephalomyelitis virus. The prevalence of the cases among horses and human beings is given in Figure 3. The median date of reported deaths among horses occurred during the week ending August 27, or two weeks in advance of the median date of onset for cases in human beings. Apparently, the peak of the outbreak among horses preceded that of the outbreak among human beings by an interval of over two weeks. Although the prevalence of the disease was much greater among horses than among human beings, the rise and fall of the outbreak in these groups was similar. The epizootic began slowly without increase for four weeks, and then rapidly reached its peak in two weeks. Remaining at this peak level for about a week, the outbreak subsided more slowly than it had begun. The last case was reported late in October, sixteen weeks after the beginning of the outbreak.

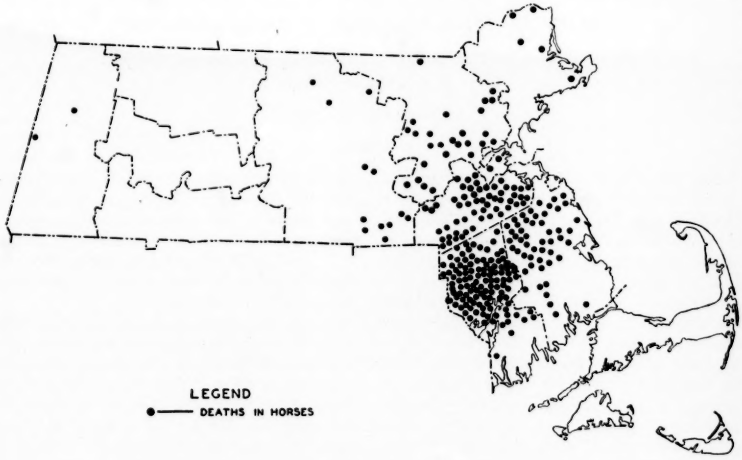

FIGURE 1. *Distribution of Equine Encephalomyelitis in Horses in Massachusetts, 1938. The deaths were most numerous in Bristol, Plymouth and Norfolk counties, the three counties between Boston on the north and Buzzards Bay on the south.*

There were no multiple cases among families, and multiple cases on the same farm or in the same stable were rare among horses. These field observations confirmed experimental evidence that the disease is not transmitted by contact between laboratory animals.

Analysis of Cases in Man

In all, the Massachusetts Department of Public Health investigated 44 cases suspected of being equine encephalomyelitis. From 9 of the patients Fothergill[4] and Webster and Wright[2] isolated the Eastern virus. These workers established the diagnosis of equine encephalomyelitis in 10 other cases by finding neutralizing antibodies for the same virus in the blood of the patients.

Eight other cases were diagnosed on the basis of pathognomonic brain lesions at autopsy. These autopsies were performed by Wesselhoeft, Smith and Branch,[8] Farber et al.[9] and Alexander.[10] A diagnosis of equine encephalomyelitis was considered justified in 7 additional patients because of the characteristic clinical findings. These cases were fatal and were not autopsied; blood specimens for neutralization tests were not obtained. Ten other cases were suspected of being due to the Eastern virus, but investigation revealed that a diagnosis of equine encephalomyelitis was unwarranted. In all, there were 34 patients in whom the diagnosis of infection with the Eastern virus seemed justified.

In analyzing these 34 cases, 2 groups were considered.

Proved cases. This group included the 9 cases in which the virus was isolated and the 10 in which the presence of neutralizing antibodies was proved. The finding of neutralizing antibodies has been considered proof of infection with the Eastern variety virus because these persons presented the same acute disease and the same clinical find-

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ings as those from whom the virus was isolated. Moreover, Fothergill[4] has been unable to find neutralizing antibodies in the blood of family contacts of proved cases, human or equine.

The age distribution of the proved and typical cases is given in Table 1. One quarter of these patients were less than one year of age, 70 per cent were under ten years of age, and only 15

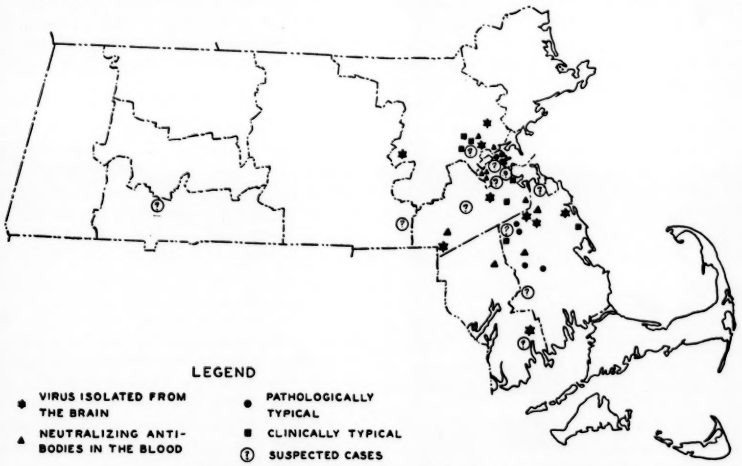

FIGURE 2. *Distribution of Equine Encephalomyelitis in Man in Massachusetts, 1938.*

In general, the distribution of human and horse infections corresponds. The apparent greater density of infections in Metropolitan Boston is due to the greater density of population.

Typical cases. The 8 cases in which the diagnoses were based on pathognomonic brain lesions, and the 7 in which there were characteristic clinical

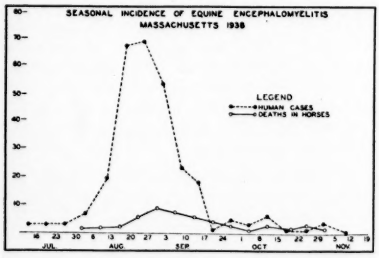

FIGURE 3. *Seasonal Incidence of Equine Encephalomyelitis in Massachusetts, 1938.*

The outbreak among horses was greater than that among human beings; the peak of the former preceded the peak of the latter by about two weeks.

findings comprise this group of patients. Since the diagnosis was less convincing, they are not included with the proved infections.

per cent were over twenty-one years of age. The sex distribution was the same in both groups: 17 patients were males, and 17 were females.

The fatality rate among the 34 patients was 74 per cent. This compares with a fatality rate of

TABLE 1. *Age Distribution.*

AGE	PROVED CASES	TYPICAL CASES	TOTAL CASES	PERCENTAGE OF TOTAL
Under 1 yr.	6	3	9	26
1–4 yr.	6	4	10	29
5–9 yr.	5	0	5	15
10–19 yr.	1	4	5	15
20–49 yr.	0	1	1	3
50 yr. and over	1	3	4	12
Totals	19	15	34	

90 per cent among horses in Massachusetts, 94 per cent in Rhode Island and 93 per cent in Connecticut.

Wesselhoeft, Smith and Branch[8] have reported the cases that were hospitalized at the Haynes Memorial in Boston. McAdams and Porter[11] described a case in Fall River, and Farber et al.[9] analyzed the cases from the Children's Hospital in Boston. The following description of the

clinical course of this disease, as observed in Massachusetts, is based on observations of the patients and on analysis of the records of all the patients.

The disease was sudden in onset, especially in children. Following a period of prodromes associated with a fever of 100 to 102°F., nausea and vomiting, and headache in older patients, there was a period of well-being, lasting from twenty-four to thirty-six hours. During this time the temperature often dropped, symptoms disappeared, and in one case a child was well enough to go to see a motion picture. As a rule, patients did not come under medical observation until the disease was well advanced, and this period of prodromes was consequently observed in only a few cases. The acute manifestations of the disease are summarized in Figure 4. The symptoms are listed in the order of frequency with which they were observed: high fever ranging from 103 to 106°F., drowsiness progressing to stupor and coma, convulsions, either clonic or tonic, a stiff neck, often developing into generalized rigidity or opisthotonos, nausea and vomiting, cyanosis, irritability in infants and headache in older patients. Twitchings were frequent and often indicated the onset of convulsions. Edema, observed in infants and small children, was not pitting, and involved the extremities and face.

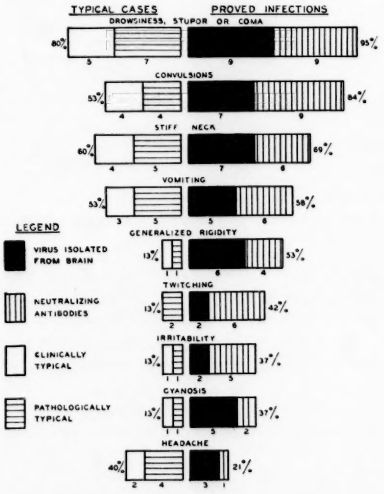

FIGURE 4. *Clinical Findings in Infections of Man with Eastern Virus of Equine Encephalomyelitis.*

The number at the end of each column indicates the percentage of patients having the symptom. The numbers under each column indicate the number of patients presenting the finding.

The duration of the disease varied from less than one day in fulminating attacks to over three weeks in the less severe forms. Table 2 gives the duration of the disease. The median length of duration was seven days for all the cases, over twenty days in the patients who survived, and four days in those who died. Of 10 patients who lived after the tenth day of illness, 4, or 40 per cent, died. The 4 patients who succumbed after twenty-one days of illness died either of exhaustion or of intercurrent infection.

TABLE 2. *Duration of Acute Illness.*

DURATION	NO. OF PATIENTS		
	LIVED	DIED	TOTAL
Less than 1 day	0	2	2
1–2 days	0	2	2
3–4 days	0	9	9
5–6 days	0	4	4
7–10 days	0	4	4
11–20 days	3	0	3
Over 21 days	6	4	10
Totals	9	25	34

A diagnosis of equine encephalomyelitis cannot be made on clinical findings alone. Laboratory data furnish confirmatory evidence. There is a leukocytosis of 14,000 to 66,000, with a differential count of 75 to 90 per cent neutrophils and 25 to 10 per cent lymphocytes. The spinal fluid is under increased pressure, and there is a pleocytosis during the acute phase, the counts averaging 500 to 1000 cells, chiefly neutrophils, per cubic millimeter. With the subsidence of the acute phase, the spinal-fluid cell count drops, and the lymphocyte becomes the predominating cell. Spinal-fluid sugar and chlorides are at a normal level, but the total protein is increased, especially in the convalescent stage.

Although blood cultures for the virus were not obtained during the epidemic, it is likely that if they are made early, using defibrinated blood, positive cultures may be obtained. Ten Broeck, Hurst and Traub[12] have isolated the virus from the experimental disease in horses, and Howitt[13] has reported the isolation of the Western variety from the blood of a patient. According to Fothergill's[4] findings, neutralizing antibodies may appear as early as the first week of the disease but are not likely to do so until the second week. Complete pathological descriptions have been written by Farber and Branch.[14]

There is no specific therapy for equine encephalomyelitis. Treatment is symptomatic and supportive. Isolation of the patient is not necessary, since the disease is not transmitted by direct contact. Chick vaccines, effective in the immunization of horses, have not yet been developed for use in man. Moreover, they are of no value in the treatment of horses.

Follow-Up of Cases

All 9 surviving patients in whose blood neutralizing antibodies to the Eastern variety of virus were found were seen at varying intervals from the date of the original illness. In all cases except one, this follow-up was made approximately a year after onset. These cases have been under the care of various physicians and clinics, and this analysis is made with the permission of each. Table 3

Table 3. *Summary of Follow-Up.*

Case	Sex	Age at Onset	Interval to Last Observation	Residual Symptoms
D. A.	M	12 mo.	13 mo.	Mental retardation and right hemiparesis
M. B.	F	6 yr.	15 mo.	None
R. C.	M	12 mo.	14 mo.	Mental deficiency, aphasia and left hemiparesis
L. F.	M	8 yr.	15 mo.	Definite mental retardation (age about 3 yr.) and partial aphasia
D. L.	F	4 mo.	13 mo.	Mental retardation, strabismus and frequent convulsions
M. C.	F	18 mo.	13 mo.	Mental deficiency, marked lack of emotional control, right hemiplegia, impaired vision and partial deafness; patient speaks only a few words.
C. D.	M	6 yr.	15 mo.	None
T. O'C.	M	4 mo.	3 mo.	None
R. R.	F	1 mo.	11 mo.	"Decerebrate animal" and mass reflexes; eyes do not react to light.

summarizes the observations in the surviving patients. All but 3 developed disabling sequelae. Six showed evidence of cerebral degeneration, with definite mental retardation. Three patients continued to suffer from a hemiparesis, 3 from complete or partial aphasia, and 1 from emotional instability.

At this early date it is impossible to prognosticate the eventual course of these sequelae. However, it seems not unlikely that they are permanent and may even be progressive. In encephalitis lethargica, sequelae have appeared as late as five or more years after the original illness, and it is therefore important to follow these patients for at least five years.

Mosquito Survey

As soon as the true nature of the equine-encephalomyelitis outbreak was realized, the Massachusetts Department of Public Health, with the assistance of the United States Public Health Service, undertook the organization of a mosquito survey. During August and September, 1938, mosquitoes were unusually prevalent. On September 21, a severe hurricane swept over New England and many mosquitoes were destroyed. Identification of specimens collected in October revealed Culex and Anopheles mosquitoes. Aedes were no longer active, and relatively few were collected.

The study of the history of equine encephalomyelitis reveals that the disease often recurs in the same region in succeeding years. Therefore, during the early months of 1939 the Massachusetts Department of Public Health, under the direction of the commissioner, Dr. Paul J. Jakmauh, began to make plans for a state-wide mosquito survey. The aims were to ascertain whether mosquitoes are the natural vectors of the disease, to find naturally infected mosquitoes, and to ascertain the distribution of experimentally demonstrated vectors, so that when and if the disease recurred, control measures might be effectively and efficiently instituted.

The active work of the survey was made on a state-wide basis during the months of July, August, September and October. It was carried out in co-operation with the Work Projects Administration, which supplied a personnel of 150. This personnel was under the technical direction of a member of the Department of Public Health and ten entomologists, who were employed during the course of the survey. Almost 50,000 separate collections of mosquitoes comprising 278,000 specimens were made by the WPA personnel and volunteer collectors, who were obtained through the co-operation of the local boards of health. Various state and federal institutions, schools, summer camps and many other agencies made numerous collections of mosquitoes. It is the purpose of this paper to make a report on this mosquito survey so far as it is concerned with equine encephalomyelitis.

In all, 56 different species of mosquitoes were collected in Massachusetts. Thirty-seven of these are biting and the remainder are nonbiting. Only 6 of the biting species have been shown by investigators to transmit experimentally the Eastern virus of equine encephalomyelitis to laboratory animals. Positive experimental transmission has been carried out with the following Massachusetts mosquitoes by Davis[15]: *Aedes cantator, A. sollicitans, A. vexans, A. atropalpus* and *A. triseriatus. A. taeniorhynchus* was not collected in numbers large enough to conduct transmission experiments; however, Ten Broeck and Merrill[16] have demonstrated

that it is a laboratory vector of the Eastern variety of virus. Culex, Anopheles, Mansonia, Uranotaenia, Wyeomyia and other genera of mosquitoes have thus far been unable to transmit the disease to laboratory animals in experiments conducted by Davis and other investigators.

Data, collected by the survey, have furnished additional epidemiological support to the mosquito-transmission theory of equine encephalomyelitis.

Seasonal prevalence of vector and disease. For the effective transmission of a mosquito-borne disease, there must be a certain minimal numerical relation between the vector, host and susceptibles. When the vectors are unusually prevalent, conditions for increased transmission are present. In 1938, the outbreak reached a peak in late August and early September. In 1939, the seasonal prevalence of mosquitoes was determined. Whereas the genus Aedes was more abundant in spring and early summer, the vectors, *A. atropalpus, A. cantator, A. sollicitans, A. taeniorhynchus, A. triseriatus* and *A. vexans*, were most numerous in August and September. The seasonal prevalence of the suspected vector corresponds to that of the disease. Therefore, conditions most conducive to the transmission of the virus exist in the late summer and early autumn. The unusual abundance of mosquitoes in 1938 provided optimal conditions for the outbreak.

Geographical distribution. The vectors of equine encephalomyelitis were present in those areas where, formerly, the disease was present. Only two vectors, *A. vexans* and *A. triseriatus*, were present in all places where cases occurred. *A. triseriatus*, however, was found to be only one fifth to one fifteenth as numerous as *A. vexans*. Therefore, statistically, it seems that *A. vexans* is the most important vector of equine encephalomyelitis.

The geographical distribution of the vectors of equine encephalomyelitis is shown in Figure 5. *A. vexans* and *A. triseriatus* are state-wide in distribution. *A. sollicitans* and *A. cantator* are both salt-marsh mosquitoes, which are usually limited to within ten miles of the salt water, but which in exceptional circumstances may be found fifteen miles inland. *A. atropalpus* breeds in clear water that has accumulated in eroded rock formations, as in the Connecticut Valley, Essex County and Cape Cod. *A. taeniorhynchus*, which Ten Broeck and Merrill[16] have shown to be able to transmit the Eastern variety virus experimentally to laboratory animals, has been collected only along the southern coast of Massachusetts — on the island of Martha's Vineyard and adjacent Cape Cod.

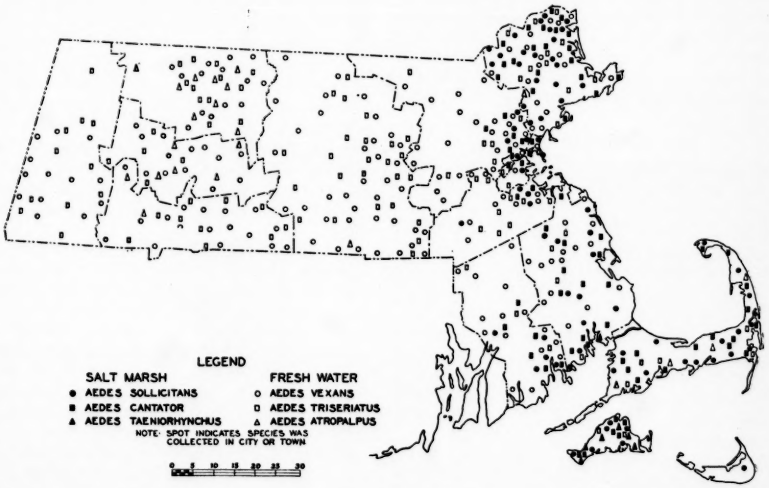

FIGURE 5. *Distribution of Vectors of Equine Encephalomyelitis (Eastern Variety) in Massachusetts, 1939.*

Aedes vexans *was 5 to 15 times as numerous as* A. triseriatus. *The salt-marsh species were limited to an area fifteen miles from the nearest breeding place.*

The most probable natural vector. Further confirmatory evidence that *A. vexans* is the principal vector is found in the distribution of equine-encephalomyelitis cases in 1939. Twelve cases in horses were diagnosed by the Massachusetts Department of Agriculture. Although most of these were diagnosed on clinical findings, the virus was isolated from the brains of two horses that had lived and died in the towns of New Braintree and Westborough. These towns are so far inland that no salt-marsh mosquitoes were collected, and the probability that a mosquito of this group infected these horses is negligible. The nearest breeding place of the salt-marsh vector was twenty miles from Westborough and over fifty miles from New Braintree. Fifteen miles away from their breeding places is the greatest distance at which the adult salt-marsh mosquitoes, *A. sollicitans* and *A. cantator*, were collected. When wind-borne, both species have been recorded to fly farther. However, the prevailing winds in the summer and early autumn blow from the west and southwest, hence the salt-marsh mosquitoes would have to fly against the wind for twenty to fifty miles to reach these two towns. Furthermore, long flights of salt-marsh mosquitoes are a rare phenomenon; statistically, therefore, the chances that a few adults may survive such long flights are almost negligible. All these facts point to a mosquito other than a salt-marsh Aedes as the carrier. Only two vectors were found in this area, *A. triseriatus* and *A. vexans*. Since *A. vexans* is five to fifteen times as numerous as the former, statistically, *A. vexans* is the most probable vector responsible for the infection of these horses.

Age distribution of cases according to the bionomics of vectors. The determination of the bionomics of the vectors of equine encephalomyelitis offers explanations of several phenomena observed in the 1938 outbreak. Twenty-six per cent of the cases in human beings were in infants under one year of age, and 70 per cent of the patients were children under ten. Although detailed information about individual cases has not been obtained, the high incidence among the young age groups may be explained by the following observations. During the summer, infants are often left to sleep outdoors, sometimes without protective netting, and are unable to defend themselves against mosquitoes. Moreover, children under ten who play outdoors the greater part of the day are less efficient than older groups in protecting themselves, since they do not react to a mosquito until it has bitten them. Thus infants and children who sleep or play outdoors are more apt to be bitten than older persons who, taught by experience, react to the buzzing or alighting of a mosquito.

Outdoor vectors. It was observed that horses which were kept outdoors formed the larger portion of the cases. Data collected during the survey indicate that the vectors are more likely to bite in the open than indoors. Hence, horses that are outdoors are more apt to be infected.

Of all mosquitoes captured on man outdoors, 60 per cent were vectors, whereas only 6 per cent of those caught indoors were carriers. Therefore, the chances that a mosquito is a vector are ten times as great if the biting occurs outdoors than if it occurs indoors. These findings suggest why infants and children comprise such a large number of the cases.

Summary

Equine encephalomyelitis due to the Eastern variety virus infected 34 persons during an outbreak of this disease among horses in southeastern Massachusetts in 1938. Small infants and children under ten years of age constituted 70 per cent of the cases.

The onset of the disease is acute, the course rapid, and death occurs in less than ten days in the majority of cases.

Permanent sequelae occurred in 6 of the nine surviving patients. The sequelae, in the order of frequency, were mental retardation, hemiparesis, aphasia and emotional instability.

Three salt-marsh species — *Aedes cantator*, *A. sollicitans* and *A. taeniorhynchus*, and three fresh-water species, *A. atropalpus*, *A. triseriatus* and *A. vexans*, all vectors of equine encephalomyelitis — were collected by the survey in 1939. The former are usually limited to within ten miles of salt water. Two, the fresh-water vectors *A. triseriatus* and *A. vexans*, were state-wide, the latter being five to fifteen times as prevalent as the former.

A. vexans is probably the most important vector of equine encephalomyelitis in Massachusetts. Along the coast the *A. sollicitans* and *A. cantator* are about as numerous as *A. vexans* and may have been, in part, responsible for the outbreak of 1938 in the coastal region.

The epidemiologic and entomologic data collected by this survey support the laboratory evidence that equine encephalomyelitis is transmitted by mosquitoes, because the geographical distribution and the seasonal prevalence of the disease and the vectors have been found to be the same. The biting habits of the vectors of equine encephalomyelitis suggest that man and animal outdoors are exposed ten times as much to the chance of infection as those in buildings.

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A FIERY ORDEAL*

JOHN FLEET, M.B.

BOSTON

I GRADUATED from Harvard College in 1785 and determined to become a doctor. I at once entered the new Medical School at the College in Cambridge, and for three years sat under those erudite young teachers, Dr. John Warren, Dr. Benjamin Waterhouse and Dr. Aaron Dexter. On May 23rd, 1788, shortly before I obtained my degree of Bachelor in Medicine at Commencement, I appeared before the Censors of the Massachusetts Medical Society in the County Court House in Boston, where meetings of the Society were held, hoping on their approval to obtain a license to practise the healing art in the Commonwealth.

The Board of Censors of the Society at the time was made up of Dr. James Lloyd, Dr. Joseph Gardner, Dr. Isaac Rand, and Dr. Samuel Danforth of Boston; and Dr. Oliver Prescott of Groton. Dr. Prescott, being hard of hearing, took no particular part in my examination; business prevented Dr. Danforth's attendance.

Dr. Lloyd was the senior member present. He ranked high in the profession in those days. He had taken the lead in regard to the practice of surgery in Boston and was the first who introduced hereabouts the male practice of the obstetric art as a general appendage to the attire of a physician. He was very successful in it and consequently was greatly esteemed among the ladies. He entertained a great deal of company, kept a genteel equipage and a suite of servants; his horses were esteemed equal to any in the town. In brief, he was a gentleman of the old stamp and deservedly respected and valued.

Dr. Joseph Gardner was employed both as a physician and surgeon as much as any other gentleman in the profession. He, too, was an older man, in practice for some years, and included among his pupils John Homans and Charles Jarvis. He pretended that he looked upon learning as superfluous; that the bedside was the only school for a physician. But he did study and was a more learned man than he chose to appear. When he wished he could be both witty and satirical.

Dr. Isaac Rand, the youngest Censor, was a pupil of Dr. Lloyd's. He acted as Secretary of the Board. He was interested in mathematics and was always seeking for something like scientific fact on which to lean in his profession. For want of knowledge he was continually dissatisfied and read more books than any physician in Boston. To be sure, he was apt to pin his faith in the last book he read: but he was a successful practitioner, had a discriminating judgment, and was remarkably neat in his operations.

These were the gentlemen who were to examine me. The examination was to be no easy task because Dr. Warren and his new school at Harvard were by no means popular with physicians of the Massachusetts Medical Society. Dr. Warren was regarded by many as an artful man who had got to the windward of his colleagues in establishing a medical school and himself a Professor; Dr. Waterhouse, in the Theory and Practice of Physic, was thought to have received his appointment in part, at least, because he was a relative and pupil of Dr. Fothergill of London: and it was common gossip that Dr. Fothergill intended to help in the development of a Medical Institution in New Eng-

*An unpublished manuscript. Submitted by Dr. Reginald Fitz.

land where his relative and student proposed to dwell; Dr. Dexter, a respectable figure, pretended to no greater knowledge of Chymistry and the Materia Medica than did several other members of the Society who also had academical leanings, so that the justification of his choice was queried.

I knew, as first product of this new School to be examined by the Censors of the Massachusetts Medical Society, that I would be thoroughly sifted. And indeed I was.

Dr. Rand began my examination by asking me to describe the process of deglutition and digestion.

I answered, "The food is first taken into the mouth, divided by the teeth, and pushed backwards by the muscles of the throat."

"Is there anything to prevent it from passing into the trachea?"

"The glottis." And then I added immediately, "The epiglottis, I mean, that covers the glottis."

"What is the epiglottis?"

"A cartilaginous substance."

"Proceed in the process," said Dr. Rand.

"The food is pushed into the esophagus, passes down to the stomach, is then mixed with the gastric juice and operated upon by the stomach. If vegetable food be used, a fermentation takes place."

"What is the upper orifice of the stomach called?"

"Cardia."

"What the other?"

"Pylorus. The food is next protruded into the duodenum, and there it mixes with the pancreatic juice and bile."

"What conveys the bile into the duodenum?"

"The ductus communis choledochus."

"Does the hepatic duct enter the ductus communis choledochus?"

"Almost always," I answered.

"Proceed with the food," Dr. Rand demanded. "The food being mixed with the bile and pancreatic juice, of what use is the bile?"

I hesitated. He repeated the question.

"It assists to form chyle," I answered.

"Go on."

"When chyle gets into the small intestines it is absorbed by the lacteals."

"What are the lacteals?"

"Vessels that convey chyle to the receptaculum chyli."

Dr. Gardner interrupted. "Where does it lie?" he asked.

"I do not exactly remember upon which vertebra of the loins," I replied.

Dr. Rand resumed the questioning. "What does the chyle pass through in going to the receptaculum chyli?" said he.

"Some small glands," I replied.

"That is not what I mean. Is there nothing else?"

"The mesentery, do you mean, Sir?"

"Yes," he said. "Where does the chyle go after it enters the receptaculum chyli?"

"It passes up by the thoracic duct to the left subclavian vein and mixes with the blood, from thence it passes with the blood into the right auricle of the heart."

"What pushes it to the heart?"

"Its own contraction and the pulsation of the aorta: the thoracic duct also has valves."

"What prevents the chyle regurgitating when it enters the vein?"

"There is a valve that prevents its return."

"Proceed in the circulation."

"The blood is propelled from the right ventricle into the lungs."

"By what vessel?"

"The pulmonary artery."

"What peculiar structure has the pulmonary artery?"

"It is said to form spiral lines around the extremities of the air vessels."

There was some confusion in a question asked here; in other words, there were several statements made. One question I recollect was what effect respiration had on these spiral lines.

"They are compressed by inspiration," I replied.

"Is the blood quickened by respiration?" asked Dr. Rand.

"Yes."

"How is the blood brought back from the lungs?"

"By the pulmonary vein into the left auricle, next into the ventricle, from thence it is protruded into the aorta and conveyed throughout the body. The aorta ascendens sends off the carotids and subclavians, the right carotid from the subclavian, and left from the aorta itself."

"What arteries convey the blood to the head?"

"The carotids and vertebrals."

"How do they convey blood for secretion to the brain: by the dura mater or into the substance of the brain itself?"

"They are spread on the dura mater and pia mater, pass with it into the convolutions of the brain and into the cortex, and if there is any secretion they perform it."

Dr. Rand now said, "Give us some account of the veins of the brain."

"They return the blood into the sinuses."

"Is there any peculiarity in the veins of the brain?"

"Sinuses are peculiar to the veins of the brain."

"Where does the blood go next?"

"The blood passes from the longitudinal sinus to the lateral ones, from thence to the base of the brain, and goes out at the jugular veins."

"What arteries enter the large foramen of the os occipitis?"

"The vertebral. They pass along a notch or hole in the vertebrae of the neck."

"How many lobes has the cerebrum?"

"Four."

"How many has the cerebellum?"

"I believe it consists but of one."

"What is the junction of the cerebrum and cerebellum called?"

"The medulla oblongata."

"What is it called afterwards?"

"Spinalis."

"What prevents one hemisphere of the brain from falling upon the other?"

"The processus falciformis."

"Where does it arise?"

"From the crista galli and proceeds along the sagittal suture."

"What are the lymphatics of the brain, is there any other system of vessels, and do the arteries terminate in them?"

"No, Sir," I answered. "The lymphatics arise from cavities, interstices of fibres and the parenchyma."

"Where do the lymphatics of the lower extremities go? Suppose a wound in a lymphatic and a virulent matter introduced, where will it appear?"

"In the groin."

"Right," said Dr. Rand. "Where do they go next?"

"They pass into the receptaculum chyli, and mix with the chyle."

"Do they come from all parts of the body?"

"Yes."

"Name the bones of the head."

"The ossa parietalia joined together by the sagittal suture, os frontis joined to the parietalia by the coronal suture, ossa temporalia by the squamous sutures, os occipitis by the lambdoidal suture, os sphenoidis by the squamous and transverse sutures and the os ethmoides."

"What bones form the orbit?"

"Ossa frontis, palatia, maxilla superioris, os planum, os unguis."

"The bones of the face?"

"I believe there are thirteen," I answered. "I have enumerated some of them."

"Proceed with the rest."

"Ossa nasi, ossa spongiosa, superior and inferior vomer, maxilla superior and inferior."

"You have omitted ossa malarium," said Dr. Rand rather coldly. "Now give the bones of the arm."

"Os humeri, radius, ulna, carpus, metacarpus, phalanges."

"What prevents the shoulders falling in?"

"Clavicula," I answered.

"To what are they joined?"

"To the sternum and scapula."

"What process has the scapula and what is its use?"

I did not readily recollect the name and he told me. Then he asked, "What are glands and their composition?"

"They are composed of arteries, veins, and excretory ducts."

"Do arteries terminate any other way?"

"They open into cavities and on the surface of the body."

"What kind of articulation is the joint of the elbow?"

"Ginglymus," I replied.

"What is ginglymus?"

"It is an articulation resembling a hinge."

"Name another instance."

I did not answer immediately. He asked what kind of joint that of the ribs was. I did not know. He said that was ginglymus.

Then he asked, "What is it blood is composed of?"

I answered, "Salt, water . . . ."

He stopped me, saying that he did not want the chymical properties but the spontaneous divisions. So I replied, "Coagulum and serum."

Dr. Lloyd now interrupted and said that he supposed I knew but that I did not recollect the proper term. Dr. Rand said that I meant crassamentum, and that there was another part, too,—coagulable lymph,—which had not been mentioned. I told him this element required an operation to be recognized but he answered that it needed only a little whipping.

At about this juncture Dr. Rand was asked by the other gentlemen to desist as time was failing. So he said, "It is your turn now, Dr. Gardner, to examine the candidate in the practise of physic." After this, Dr. Rand took out his watch every few minutes and complained constantly as long as the examination lasted that time would fail unless they made haste.

Dr. Gardner began with, "What is inflammation?"

I replied, "An increased action of the vessels of any part."

"What are the causes?"

"Anything that irritates or excites the arterial system to greater motion."

"Well, suppose a man had an inflammation in his leg, what would you do?"

I made some pause until he went on, "I mean a phlegmon, I wish to be understood. What would you do?"

"I would first endeavor to discuss or resolve it."

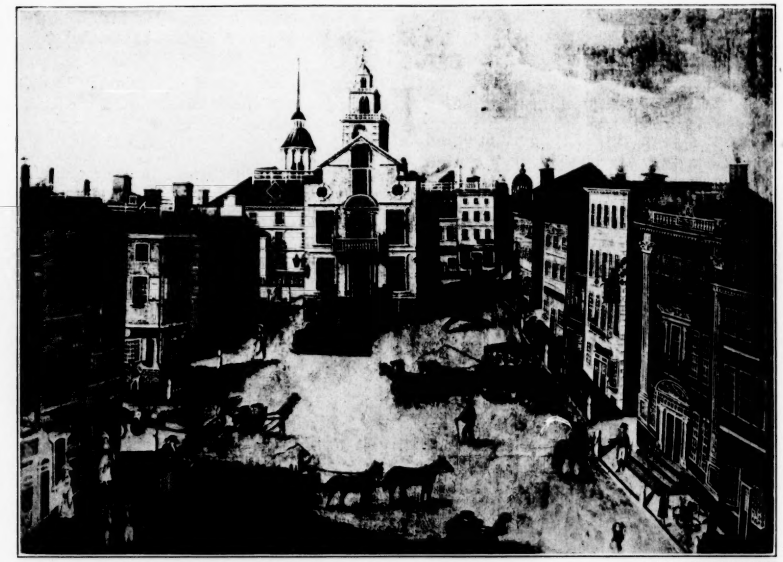
"What are the terminations of inflammation?"

"The common terminations of inflammation are in resolution, suppuration, gangrene or mortification."

Dr. Rand interrupted. "How about fomentations?" I said I would use them also. Dr. Gardner went on. "What do you mean by emollients?"

"Such things as will take off the stricture of the vessels and allow of an effusion from them."

"What is a pleurisy? How do you know a person has got the pleurisy?"



MASSACHUSETTS HISTORICAL SOCIETY

FIGURE 1. *The Massachusetts Medical Society in 1788.*

The Council met in the State House; the censors met in the Court House on Queen Street, the building on the right with the cupola.

"By what means would you resolve the inflammation?"

"By allaying the increased action of the vessels."

"How?"

"By astringents and sedatives."

"What kind?"

"Sometimes cold water is used, or saturnine applications."

"Suppose you could not resolve it?"

"If after endeavoring all in my power to resolve the tumor I should find it could not be done, I would bring it to suppurate."

"To maturate, yes," said Dr. Gardner. "Well, by what means?"

"By emollient cataplasms."

"Pleurisy is known by a pain in the side, tightness over the chest, a cough, a fever."

"What are remedies to be used?"

"The chief remedy is blood letting."

"How soon would you bleed a second time?"

"If the pain of the side was great and respiration greatly obstructed, I would repeat the bleeding in a few hours."

Dr. Rand interrupted by asking how much blood a man could afford to lose. I replied, "I think I have seen an author who says we may take away as much as three or four pounds."

"Did you ever read Sydenham?" asked Dr. Rand.

"No," I answered.

"You ought to," replied Dr. Rand. "He says about forty-two ounces." But the other examiners all then said much more was frequently taken. After this interruption, Dr. Gardner continued, "What else would you do?"

"I would blister the side and if the patient would bear no more bleeding and symptoms continued violent, I would cup and scarify the side and draw some blood that way."

"Would you do anything else?"

"I would use demulcents for the cough."

"What demulcents?"

"Flaxseed tea and such things," I answered. "I would also use diluents and nauseating doses to determine to the surface and relieve the extreme vessels. I would keep the body open and soluble."

Dr. Rand now interrupted by asking, "What is a bilious fever?" I was considering what description it would bear when he said it was a typhus he meant, which rendered the matter more obscure. I told him a bilious fever arose from acrid bile being absorbed. Dr. Rand asked, "What is the predisponent cause?" I could not tell; for I could not reconcile bilious fever and typhus being the same thing. Dr. Rand asked me if the season was not the predisponent cause and what season typhus happened in. I said, "Toward the close of summer." "Do you not mean in the autumn?" then said he. I told him I supposed heat was an occasional cause.

"No," said he. "What is the cure?"

"Evacuants, diluents and refrigerants," I replied.

Dr. Gardner asked what I meant by diluents. I replied, "Water and teas that contain a great deal of water."

It was now Dr. Lloyd's turn to question me in Surgery. Said he, "You have seen amputations. Describe one, for instance below the knee."

I answered, "I would place the patient on a table of a convenient height."

"Stop!" said he. "Where are your dressings?" I told him I thought it unnecessary at this time to describe such minutiae. He went into a pathetic encomium on the wisdom and ingenuity of a surgeon who should have the foresight and address to prepare every article that should be necessary. So I enumerated double-headed roller, needles, tenaculum, great knife, cutting saw and retractor.

"Where is your lint?" asked Dr. Lloyd, "and would you not have something of a pledget and a cross bandage?" I enumerated these various articles and was about to describe how I would place them all in a convenient dish. Here I was so interrupted by proposals, repetitions and so many questions that I was confounded and held my tongue.

Dr. Gardner said, "Take time, my dear, we don't mean to catch you."

At last I got the patient on the table again.

"How high a table?" asked Dr. Lloyd.

I answered, "One of convenient height — about two and a half feet high."

I believe that he said three feet high, and then turning to the others said, "That is the proper height."

At length I had a chance to continue. "I would secure him by two assistants, and place the tourniquet on the artery."

"Where?"

"Toward the groin," I said, "where I could feel the vessel beat. I would have an assistant to command the tourniquet, one to draw up the skin, and another to support the leg. I would then take the great knife and desire the assistant to retract the skin a little, making the incision a hand's breadth below the knee."

Dr. Gardner asked me to point where, and I pointed to the proper place. Dr. Rand said, "Of course you chose this point to avoid the tendon of the patella," and I answered, "Yes." I then went on, "After I had made the incision through the cellular membrane I would beg the assistant to retract the skin and cellular substance a little, and would make the second incision as high as possible and down to the bone."

"Straight?"

"Yes." I was going to apply the retractor when Dr. Rand asked, "Where is your catlin? If there is a portion of muscle undivided between the bones the catlin must be used." This interruption made me forget the retractor and they said nothing about it, so I continued. "I would then take the saw and saw through both bones and take the limb away. After clearing away the blood I would ask the assistant to loosen the tourniquet; whatever arteries I could find I would draw out with the tenaculum and have an assistant to tie them, repeatedly requesting the person who commanded the tourniquet to loosen it that I might discover the arteries."

Dr. Rand interjected another question. "Would you not wipe away any clotted blood with a sponge?"

"Yes."

"What would you do next?"

"I should draw the lips of the wound as near together as possible over the bones and secure them by ligatures."

"By needles do you mean?" asked Dr. Rand.

Undesignedly I said, "Yes," and immediately he said he should not think of it unless the lips of the wound were flabby. While he was saying this I saw the error I was led into and interrupted him. "I would secure them with sticking plaister," I said, "and then I would apply a pledget of some emollient ointment."

"No lint?"

"No," I answered. They then consulted together and concluded that lint was necessary. At length I continued. "I would place on the cross bandage and apply the roller."

"How long should the bandage be?" asked Dr. Lloyd.

"Four yards would bring it round the thigh and secure it."

"Would you not secure it to something passed around the waist?"

"Yes. I would then get the patient to bed."

Dr. Gardner asked me what to do afterwards. I said I would bleed if the pulse was hard and full. Dr. Rand asked what should be done if the patient was irritable. I said, "If the pulse was low and the patient exhausted, I would give him bark and wine."

Their frequent complaint of want of time and my expectation of being asked many more questions induced me to answer much quicker than prudence otherwise directed. But I recollect no other questions in surgery and I think I am able positively to say there was no other of any sort. Drs. Rand and Gardner seemed by their motion to be done.

Dr. Lloyd now drew out of his pocket a paper and said he had some more questions to ask me.

"What are the dimensions of the pelvis?"

I replied, "Shall I mention bones first?"

"No, it is no matter."

"The shortest diameter of the pelvis, from the base of the sacrum to the pubes, is about four inches and a half. From one ilia to the other, or from one side to the other side of the brim, five and a half inches."

Dr. Lloyd said from side to side it was called, and Dr. Rand asked if I meant from the spines to the ilia: I replied I meant the brim of the true pelvis.

"How is labor brought on?"

"About the ninth month; from the largeness and strength of the child, the uterus is excited to contraction, the abdominal muscles are brought into action by sympathy and thus are the throes of the woman produced."

"How are labors divided?"

"Into natural and preternatural labors."

"What is to be done in a natural labor?"

"We must do nothing but wait till nature expels the child."

"Would you not set by the woman?"

"Yes, Sir," I answered, "and feel now and then if the child be in the right position."

Dr. Lloyd warned me that if I did not, I would soon lose all my business. Then he entered into another descant on the prudence of preparatory measures. I said I thought he only wanted me to give the fundamental principles. Dr. Rand interrupted by saying, "Would you not support the perineum?"

"Yes," I explained, "What I meant by doing nothing is that we should use no violence."

"You would receive the child, I suppose?"

"After the child was born I would tie the funis at about three fingers' breadth from the navel, and cut it at nearly the same distance from the ligature. I would twist the navel string round the fingers of my left hand and introducing my right would take hold of the cord higher up. When the pains of the mother came on, I would pull down by the funis gradually: at first in the direction of the coccyx, and moving it backwards and forwards would at last disengage the placenta."

"Suppose the placenta will not come?"

"It is customary to desire the patient to blow in the fist, to cough and the like."

"Suppose too there is a flooding?"

"I would extract the placenta as soon as possible; especially if the woman show any signs of deliquium."

"Suppose the placenta still would not come?"

"I would introduce my hand into the womb and endeavor to separate it."

Now Dr. Rand said, "Suppose the woman faint and the flooding stopped, would you now think of extracting the placenta?" I am not certain how I answered this question.

"What is a laborious labor?" continued Dr. Lloyd.

"A laborious labor is when the child cannot be delivered without the use of instruments."

Dr. Rand said he believed I was mistaken: a laborious labor was when the head was large and the pelvis small or rigid. I told him that Orme called such labor a lingering labor; but when recourse was to be had to instruments he called the labor laborious.

"Give us an instance of preternatural labor; how to deliver a breech or, let us say, an arm presentation."

"When the arm presents and the os uteri is sufficiently dilated, break the membrane and push

up the body, at the same time endeavor to get the feet; having got them extract by the feet."

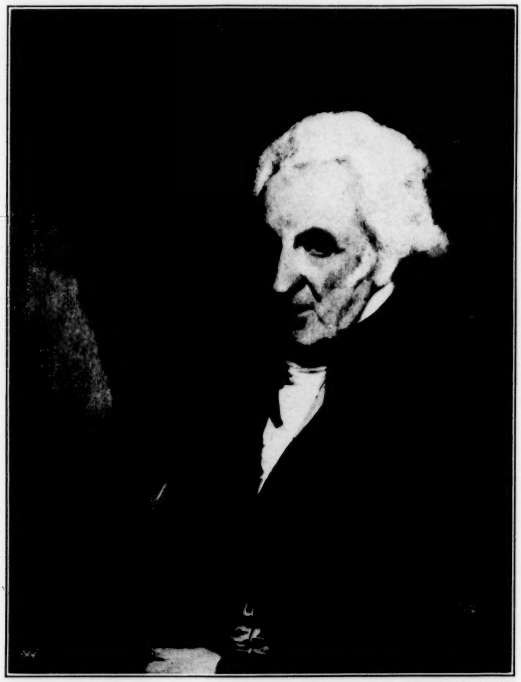
"How is it in the case of twins?" asked Dr. Lloyd.

"The twin case is merely like the others."

"When one child is born what do you do?"

"Suppose the placenta adheres to the os uteri when labor comes on, or when by introducing your finger in the os uteri you can feel nothing but the placenta, what is to be done?" asked Dr. Lloyd.

"I would separate the adhesion and deliver the child as soon as possible."



BOSTON MEDICAL LIBRARY

FIGURE 2. *Dr. James Lloyd.*

A leading surgeon and obstetrician of Boston and a conscientious censor of the Massachusetts Medical Society.

"Wait for the descent of the other."

"If," said Dr. Rand, "after the first child be born and you have cut the navel string, and after the first gush of blood is over bleeding should continue, what would you think of?"

"I should suspect another child and would secure the funis and wait for the other child."

"Should you not extract the placenta?"

"No," I answered.

"Suppose the other should not readily come?"

"After some time I would feel for it and extract it."

"Suppose during this time the woman floods or bleeds to death?"

"I know of no way to prevent it."

"Did you ever read of such a case?" said Dr. Rand.

"I do not recollect. Dr. Warren told me once of having such a case."

Dr. Rand once again interjected his ideas. He asked me if I had ever examined a placenta. If I had, he said, I would know that it is divided into lobes and that these were connected together by cellular membrane. "In this last case, these lobes

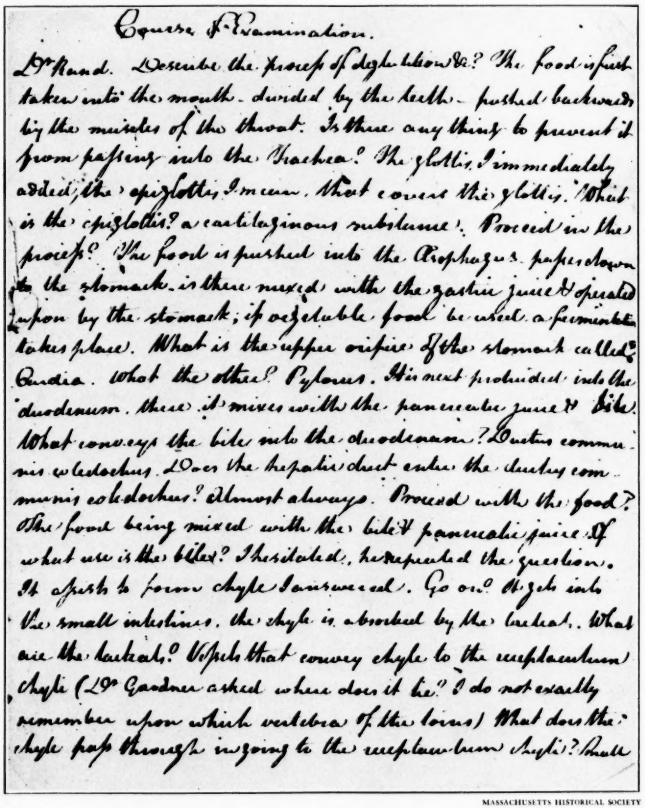
should be divided by the finger and the cellular membrane should be separated: this may be done without effusion of blood." I asked the doctors if cases of this character were not generally fatal. They all answered, "By no means."

"How do you distinguish pregnancy from amenorrhoea?" finally inquired Dr. Lloyd. Before I had a chance to reply, Dr. Gardner got up and said with some exclamation, "Don't ask him that question for I never knew myself!"

The question subsided and I was dismissed. After this ordeal, I realized that all medical knowledge was not shut up in the brains of my professors.

* * *

John Fleet was a member of the first class to graduate in medicine from the Harvard Medical School and was the first assistant appointed in the Department of Medicine. He was the first librarian of the Boston Medical Library.



Course of Examination.

Dr Rand. Describe the process of deglutition &c? The food is first taken into the mouth - divided by the teeth - pushed backwards by the muscles of the throat. Is there any thing to prevent it from passing into the Trachea? The glottis. Immediately added, the epiglottis, I mean, that covers the glottis. What is the epiglottis? a cartilaginous substance. Proceed in the process? The food is pushed into the Oesophagus - passes down to the stomach - is there mixed with the gastric juice & operated upon by the stomach; if vegetable food be used a fermentation takes place. What is the upper orifice of the stomach called? Cardia. What the other? Pylorus. Is next introduced into the duodenum. There it mixes with the pancreatic juice & bile. What conveys the bile into the duodenum? Ductus communis choledochus. Does the hepatic duct enter the ductus communis choledochus? Almost always. Proceed with the food? The food being mixed with the bile & pancreatic juice &c what are is the fæces? I hesitated, he repeated the question. It starts to form chyle I answered. Go on? Chyle into the small intestines. the chyle is absorbed by the lacteal. What are the lacteals? Vessels that convey chyle to the receptaculum Chyli (Dr Gardner asked where does it be? I do not exactly remember upon which vertebra of the loins) What does the chyle pass through in going to the receptaculum Chyli? Small

MASSACHUSETTS HISTORICAL SOCIETY

FIGURE 3. *A Page from Dr. John Fleet's Examination Notes.*

"These notes were taken the evening and day following my examination, being advised so to do before examination as a matter of satisfaction." Their accuracy was solemnly attested to before J. Brooks, justice of the peace.

He wrote his account of his examination by the censors of the Massachusetts Medical Society in the evening following the afternoon on which it was given. The manuscript was found recently in the Massachusetts Historical Society and is now printed through the courtesy of Mr. Allyn B. Forbes. It has been edited slightly by Dr. Reginald Fitz.

The censors did not consider Dr. Fleet's performance on the occasion passable. He was made to undergo re-examination a few weeks later, when he passed with flying colors.

The questions asked and the answers given serve not only as an example of an early general medical examination but also reflect fairly representative New England medical opinion in that era.

DIAPHRAGMATIC HERNIA PRESENTING THE CLINICAL PICTURE OF ACUTE COR PULMONALE*

Report of a Case

SYLVESTER MCGINN, M.D.,† AND LOUIS M. SPEAR, M.D.‡

BOSTON

THE acute cor pulmonale was originally described[1] as a sudden distention of the chambers of the right heart as a result of an obstruction to the pulmonary circulation by a pulmonary embolus. A case of acute cor pulmonale has recently been observed, however, in which interference with the pulmonary circulation was caused by a diaphragmatic hernia. The case is interesting because of the age of the patient, because it was one of the first cases of diaphragmatic hernia to be recognized by roentgenogram, and because of the similarity of some of the symptoms to those of coronary disease and diaphragmatic hernia. The recognition of these clinical conditions and the establishment of the correct diagnosis may completely alter the prognosis and may change the therapy to include lifesaving surgical measures.

Cyanosis always suggests the possibility of a cardiovascular abnormality, and the commonest cause of its acute form is pulmonary embolism followed by an acute cor pulmonale. Brewster[2] and Stoll[3] have reported 2 cases of diaphragmatic hernia, however, in which the outstanding features were cyanosis and dyspnea. Not infrequently, symptoms caused by diaphragmatic hernia have been erroneously attributed to cardiac disease. Healey[4] noted that substernal discomfort, sometimes with left shoulder pain, and regurgitation of food when the patient was in the supine position were the commonest complaints of 53 of the earliest reported cases of diaphragmatic hernia. Stobie[5] has reported that in addition to gastrointestinal symptoms, patients with this condition may have mid-epigastric distress, with pain referred to the shoulder and down the arm, palpitation, tachycardia and dyspnea. Roch[6] has discussed a case of angina pectoris that was falsely diagnosed because of symptoms associated with a diaphragmatic hernia. Electrocardiographic changes depending on the gaseous distention of a diaphragmatic hernia have been observed by Kalk and Koelsch[7] who believe that there are variations in the coronary blood flow due to vagal irritation caused by the hernia.

CASE REPORT

An 81-year-old woman entered the hospital for the third time on March 13, 1940. She had been quite well until 1919, when she was first troubled with attacks of difficult breathing and pain in the left precordium. It was thought that she did not have angina pectoris. There had been one severe attack marked by dyspnea and cyanosis that was similar to the one described in the present illness. Physical examination at that time was negative, except that the blood pressure was 180/110, and the heart was enlarged to the left.

In 1925, a secondary anemia was found, and this condition persisted throughout the rest of her life. Roentgenograms made in 1930 showed a diaphragmatic hernia (Fig. 1). A fall in 1931 resulted in a fracture of the tenth dorsal vertebra, the treatment of which was complicated by the inability of the patient to wear a cast or to be maintained in the recumbent position. A dorsal kyphosis became increasingly noticeable after this accident. In October, 1939, she was again examined for a questionable angina pectoris, and an electrocardiogram was taken (Fig. 2). She had been suffering from mid-epigastric, low substernal distress and indigestion after meals. It was concluded that these symptoms were caused by the herniation of the stomach and were not of cardiac origin.

One week before admission, the patient became overtired and remained in bed for 3 days. The liver edge was palpated below the costal border, but except for fatigue, she seemed in her usual health. On the day of admission, she was drowsy, although she could be aroused.

*From the Robert Breck Brigham Hospital.

†Instructor, Courses for Graduates, Harvard Medical School; assistant in medicine, Massachusetts General Hospital; cardiologist, Robert Breck Brigham Hospital.

‡Chief of the Medical Service, Robert Breck Brigham Hospital.

Respirations were rapid and shallow, and the lips and fingernails were very cyanotic. There was no pain at any time.

Physical examination showed an elderly woman with increased shallow respirations. Cyanosis of the nails, lips, ears and cheeks was marked, and there was a mottled blueness of the skin. The neck veins were greatly distended. The pulmonic second sound was accentuated, and was much louder than the aortic second sound. Neither of these findings was present 2 days before admission. The heart was enlarged in the region of the left ventricle, the mid-clavicular line equaling 8 cm.; the left border of dullness and maximal apex beat was in the fifth interspace, 10.5 cm. from the midsternum. The heart sounds were of good quality without significant murmurs, and were regular except for extrasystoles; the rate was 78. The blood pressure was 155/90. A few fine rales were heard at the left lung base. The liver edge was palpated three fingerbreadths below the costal margin. Varicose veins and slight pitting edema were present in both legs.

The patient's condition improved slightly when she was placed in an oxygen tent. It was observed that she was very cyanotic while the oxygen tanks were being changed. It was believed that the liver was not so enlarged as it had been on admission, but palpation was difficult owing to abdominal distention, which markedly intensified the cyanosis.

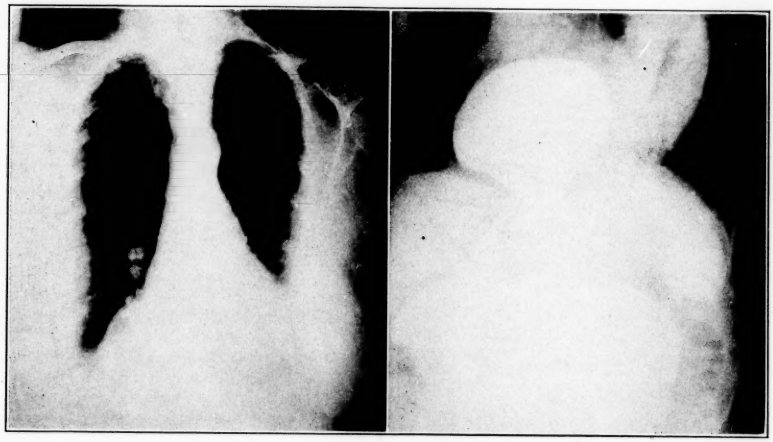
A roentgenogram of the chest was taken with a portable machine and an electrocardiogram was made; the interpretations accompany the illustrations (Figs. 3 and 4). The nonprotein nitrogen was 45 mg. per 100 cc., and the white-cell count 8650.

On the 4th day after admission, the patient was unable to take nourishment and became much weaker. The temperature rose to 103.2°F., and the pulse rate was increased to 130 at intervals throughout the day. Bronchial breathing was heard over the left lung posteriorly. The patient expired on the 5th day after admission.

Autopsy. A post-mortem examination was made by Dr. J. B. Hazard. Kyphosis of the thoracic spine was present, and dependent lividity was marked. About 200 cc. of amber fluid was found in each pleural cavity. The entire stomach, as far the pylorus, projected into the right thorax through an opening 6 cm. in diameter in the diaphragm at the esophageal hiatus. It was covered by a thin fibrous layer continuous with the diaphragmatic leaves. The left lung weighed 380 gm., the right 520, and both the right and left lower lobes were flabby, airless and, on section, purplish red. The upper lobes were grayish, hypercrepitant and easily compressed. The primary bronchi were purplish gray, and their mucosa purplish red. The branches of the pulmonary artery were wide, but the walls were of average thickness, with only occasional small yellowish plaques. No evidence of antemortem clot was found.

The heart weighed 500 gm., and the right auricle and ventricle were dilated. The right ventricular wall measured 0.8 cm. in thickness, and the left varied from 1.5 to 1.8 cm., both ventricles being enlarged. The foramen ovale was not patent, and the valves were normal. The coronary arteries were patent throughout, although there was some diminution in the size of the lumen of the left descending branch secondary to thickening of the intima and calcification.

The liver and spleen appeared to be enlarged, with



A B

FIGURE 1. *X-Ray Films Taken on June 9, 1930.*

A — *Herniation of the stomach through the diaphragm (film taken before the barium meal).*

B — *Barium-filled stomach showing the cardiac end protruding through the diaphragm of the esophageal hiatus and extending into the right thorax behind the shadow of the heart.*

approximate weights of 1800 and 300 gm. respectively, the organs not being removed. The stomach presented a purplish-gray mucosa as far as the pylorus, where it assumed a grayish tint. No evidence of necrosis of the gastric wall was found. The kidneys appeared grossly to be of average size, with a purplish-red, slightly granular surface.

Microscopic examination of the sections from the lower lobes of the lungs revealed extensive hemorrhage into the alveoli; the alveolar walls were closely approximated. The connective tissue contained an infiltration of lymphocytes. Sections from the upper lobes showed distended alveoli with thin walls. Sections from the stomach wall showed the vessels to be engorged with blood.

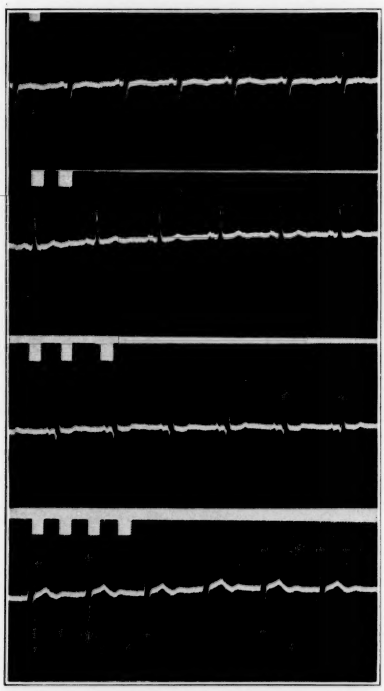


FIGURE 2. *Electrocardiogram Taken on October 25, 1939. There are normal rhythm, a rate of 70 and low T waves in all leads. The deep S wave in Lead 1 indicates a tendency to but not definite right-axis deviation. In Lead 4 the R wave is present, and there is a prominent U wave.*

Clinical diagnoses: Acute dilatation of the right side of the heart, probably due to a pulmonary embolus, diaphragmatic hernia, hypertensive heart disease and arteriosclerosis.

Anatomical diagnoses: Kyphosis of the thoracic spine, marked right ventricular hypertrophy and dilatation (cor pulmonale), left ventricular hypertrophy, pulmonary atelectasis (right and left lower lobes), pulmonary emphysema, diaphragmatic hernia of stomach into the right chest, passive hyperemia of the stomach, passive hyperemia of spleen and liver, and slight bilateral hydrothorax.

For many years the patient had a diaphragmatic hernia, apparently of congenital origin. It is of interest that despite this deformity she lived until her eighty-second year in average good health, but that the condition finally caused her death. A secondary anemia had been present for a long time, and is best explained as due to slow bleeding from hyperemic and congested vessels in the gastric mucosa, as the microscopic sections from the stomach wall suggested. This indicates that there was an interference in the blood supply to the stomach, and that there was an impairment to the venous return flow by constriction at the point of the passage of the stomach through the diaphragm. The association of a secondary anemia and a diaphragmatic hernia has been pointed out by Bock, Dulin and Brooke.[8]

The patient had a mild hypertension, which undoubtedly caused hypertrophy of the left ventricle and contributed to the cardiac enlargement. The right ventricular wall measured 0.8 cm. in thickness; this hypertrophy indicated a chronic cor pulmonale. Right ventricular strain in this case resulted from an increase in pulmonary pressure due to emphysema, which was compensatory to either or both the kyphosis and the fact that the herniated stomach was occupying space in the thoracic cavity that should have been available to the lungs. In addition to the chronic cor pulmonale, marked dilatation of the right auricle and ventricle were observed and confirmed the clinical impression of an acute cor pulmonale. The increase in the venous pressure was shown by the distention of the neck veins, and the increase in pulmonary pressure by the accentuation of the pulmonary second sound. Judging from the roentgenograms, about one third of the stomach was usually present in the chest, and apparently the entire stomach suddenly herniated through the esophageal hiatus into the right thoracic cavity. The lower lobe of the right lung and, to a lesser extent, that of the left lung were compressed and became atelectatic. This caused an acute rise in pulmonary arterial pressure, and the right heart, which was already weakened by chronic strain, became dilated and failed. Evidence of the amount of pressure exerted by the stomach on the lungs can be appreciated when it is considered that the liver was displaced downward three fingerbreadths below the costal margin. The liver edge was palpated at varying points

below the ribs, depending in all probability on the amount of air in the stomach and not on passive congestion, as we had thought before the postmortem examination. The latter examination likewise failed to demonstrate emboli in the pulmonary arteries, which we had believed responsible for the condition, even though we were fully aware that there was also a diaphragmatic hernia. The outstanding clinical signs of cyanosis and dyspnea can be readily explained by the acute cor pulmonale. It is likely that the attack of cyanosis and dyspnea in 1919 was similar to the final illness, except that the hernia reduced itself at that time.

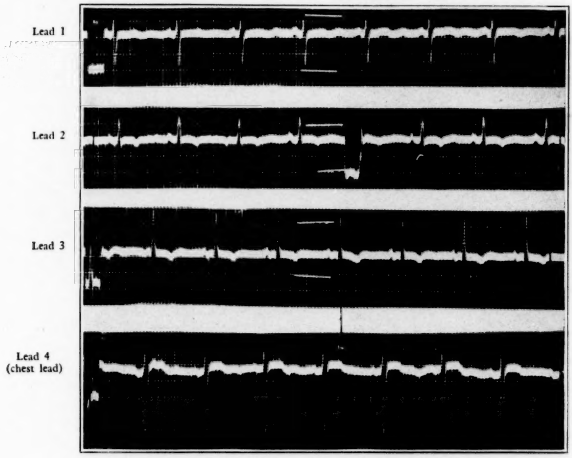


FIGURE 3. *Electrocardiogram Taken on March 14, 1940.*

There are normal rhythm, a rate of 80 and low T waves in Lead 1 (as before), but also late and deep inversion of the T waves in Leads 2 and 3 and very deep S waves in Lead 1, indicating marked right-axis deviation. In Lead 4, the R wave is present and there is high origin of the T wave. There are prominent U waves in Leads 1 and 4.

Roentgenograms made in 1930, which were the earliest we could locate, showed part of the stomach herniating through the diaphragm into the right chest. The picture made on the day after admission, four days before death, revealed that a larger portion, if not the entire stomach, had herniated through the esophageal hiatus and had replaced the usual dense shadow of the liver. The lung fields failed to show definite evidence of infarction, and the mottled dullness above the outline of the stomach was due to atelectasis. The heart shadow was enlarged at both examinations.

The electrocardiogram made on October 25, 1939, showed a prominent S wave in Lead 1, but no definite right-axis deviation. The T waves in Leads 1, 2 and 3 were low. It was believed that the tracing showed no evidence of significant coronary disease. The electrocardiogram made on March 14, 1940, the day after admission to the hospital and four days before death, differed considerably from the previous tracing. Marked right-axis deviation was present, and although the T waves in Lead 1 were still low, those in Leads 2 and 3 showed late and deep inversion. R waves were present in Lead 4, but there was high origin of the T waves. It was concluded from this electrocardiogram, together with the clinical history and the previous tracing, that an acute cor pulmonale was present.

The substernal discomfort created by a diaphragmatic hernia may readily lead to the diagnosis of angina pectoris, but the symptoms of the former condition are invariably provoked by eating and by the recumbent position rather than by exertion. The clinical picture of the present case was indistinguishable from that of an acute cor pulmonale, which we had always previously thought to be the result of an embolus in the pulmonary circulation. A roentgenogram can immediately produce evidence to differentiate these three clinical conditions; it is essential that they be diag-

nosed correctly, for the prognosis is serious in two of them, whereas patients with diaphragmatic hernias may live comfortably for many years.

If the condition illustrated by this case should be encountered again in a younger person with a more favorable operative outlook, surgical reduction and repair of the hernia would be seriously considered. Surgical discussions of this type of hernia are numerous, among them being those of Truesdale[9] and Sloan.[10] In 210 cases of diaphragmatic hernia surgically treated and reported by Harrington,[11] 195 patients were relieved of symptoms, 6 hiatus hernias recurred and 9 patients died. These data indicate definite improvement in the operative treatment of diaphragmatic hernias, chronic, traumatic or incarcerated, and justify surgical intervention, which in cases similar to the one reported may be a lifesaving procedure.

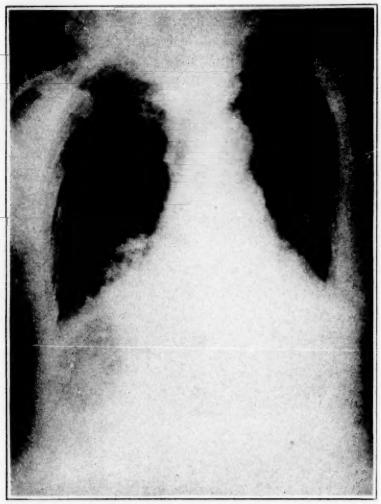


FIGURE 4. *X-Ray Film Taken on March 14, 1940.*

The greater part of the stomach occupies the lower right thorax and replaces the usual density of the liver. A mottled area is seen above the stomach shadow. The heart is enlarged and obscures the left lung base.

SUMMARY

A case of diaphragmatic hernia in an eighty-one-year-old woman is reported, including the post-mortem findings. The clinical symptoms were those of an acute cor pulmonale with marked cyanosis, dyspnea and electrocardiographic abnormalities. Compression of the lungs by an increase in the amount of stomach herniating through the diaphragm caused an acute strain on the right side of the heart. The differential diagnosis of coronary disease and diaphragmatic hernia, with possible surgical repair of the latter, is discussed.

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THE MOUTH IN HYPERPARATHYROIDISM*

Moses S. Strock, D.M.D.†

BOSTON

IT HAS long been known that careful clinical and radiographic examinations of the mouth will occasionally reveal the symptoms of systemic disease. This observation has been strikingly brought out in hyperparathyroidism. This article presents a summary of the dental findings and illustrates each oral symptom of a series of 45 cases of hyperparathyroidism. The normal oral findings should lead to a more detailed examination, and not infrequently to the recognition of hyperparathyroidism.

Knowledge of the parathyroid glands has only recently evolved. It is difficult to believe that the glands were unknown until 1880, and that tumors of the parathyroid were first discovered in 1900. It is, therefore, no wonder that the dental findings are entirely new to most observers, despite Erdheim's[1] early observations on the effect of these glands on the teeth.

The clinical syndrome of osteitis fibrosa generalisata has been known for a long time, but no great interest was excited until Mandl[2] in 1925 discovered, through the removal of an enlarged parathyroid gland, what caused the disease. Castleman and Mallory,[3] in 1934, stated that the number of cases reported at that time had reached 160. Since no investigator had reported more than 6 cases, these authors considered that the series of Albright and his co-workers,[4] which numbered 25, warranted a summary on the pathology of parathyroid disease. The Massachusetts General Hospital series has, since then, grown to 51 cases, and this report covers 45 patients.

This group of cases has been studied from the dental point of view, and the dental findings have been presented elsewhere.[4–7] The chief findings include: visible or palpable tumors of the jaw; malocclusion or distortion of the normal arrangement of the teeth (motility); cystlike cavities of the jaws; diminished dental caries; osteoporosis; closely meshed trabeculae; and absence of the lamina dura.

The case history of the famous Captain Martell[7] serves to point out the difficulties accompanying the correct diagnosis, and the post-mortem findings refute the observations of others[8] that pyorrhea or looseness of the teeth occurs with the decalcification of hyperparathyroidism. This patient was known to have had the disease for thirteen years. He had shrunk from a tall sea captain to a bent and shortened cripple. The serum calcium had ranged from 13.1 to 16.5 mg. per 100 cc., and the phosphorus from 1.4 to 3.2 mg. It required seven operations, the last an anterior mediastinotomy,[9] to locate the parathyroid tumor. The calcium and phosphorus excretions had been distinctly elevated for a long period, and the entire skeleton was markedly demineralized.

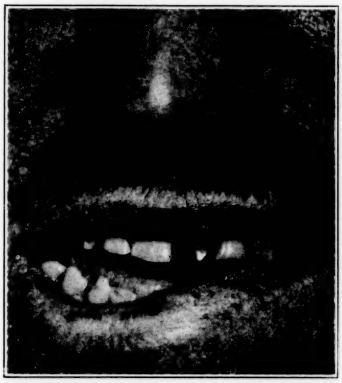

FIGURE 1. *Malocclusion or Jaw Distortion in Advanced Hyperparathyroidism.*

Note the protrusion of the lower teeth outside the upper arch. The distance was 1.5 cm. in the right lateral incisor region in this case. Because of extreme prognathism, the lower lip was hardly adequate to cover the new position of the mandible. The force of the tongue had distorted the mandibular arch. Despite the softness of the bone, the teeth were dislodged with difficulty at autopsy, and there was no evidence of periodontal disease. This intelligent patient said that seven years earlier, when his disease was known to have begun, his teeth were in normal occlusion.

When the patient's mouth was examined, the following dental symptoms were found, which were reported by Albright, Aub and Bauer.[4] The jaws were in marked malocclusion (Fig. 1). The lower jaw was prognathous to such an extent that the entire mandibular dentition was in mesial occlu-

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sion. Several visible masses distorted the normal contour of the alveolar process. By radiogram numerous punched-out areas resembling cysts could be seen (Fig. 2). The entire mandible was osteoporotic, and in sharp contrast to well-calcified teeth. The trabeculation of the mandible and maxillas was closely meshed and similar to ground glass in appearance. The lamina dura was abpleted of calcium and phosphorus that it was possible to mold the shape of the jaw by the pressure of one's fingers, yet there was no apparent lack of calcium in the teeth, clinically or by radiogram. This was more apparent at autopsy. The fingers holding the upper jaw could easily mold the maxillas to any shape. The teeth returned to their earlier position on release of pres-

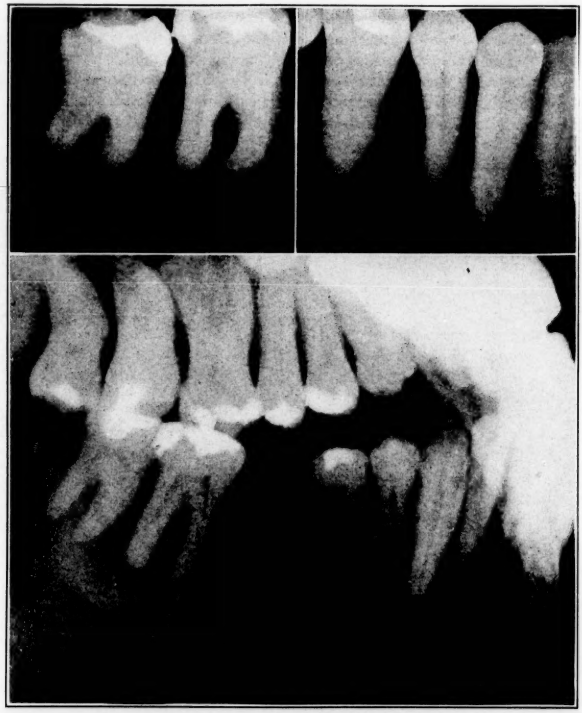

FIGURE 2. *Changes Due to Hyperparathyroidism.*
Note the lack of lamina dura, the diminished trabeculation, the remarkable contrast of the teeth against the bone, the lack of caries and the cystlike cavities.

sent from almost all the teeth. Despite this, radiographic examination showed only one or two obvious cavities. The history revealed that in the patient's numerous hospital stays for thirteen years his dental attention had been limited to one or two temporary fillings, and that his occlusion had been normal fifteen years earlier.

Here was a patient whose bones had been so desure. They were not loose, and a specimen tooth was extracted only with considerable effort.

Bauer[7] summarized the alterations in the calcium and phosphorus metabolism and said that they could be accompanied by any or all the following signs and symptoms: polydipsia, polyuria, weakness and loss of strength, constipation, loss of appetite, loss of weight, indefinite muscle and

joint aches and pains, — commonly diagnosed as "rheumatism," arthritis or neuritis, — bone tenderness, frequent fractures, — often following slight trauma, — decreased excitability of the nerves, skeletal shortening, kyphosis, bone tumors, — frequently diagnosed as epulis of the jaw or giant-cell tumor in other bones, — kidney or ureteral stones — usually bilateral — and, frequently, anemia with leukopenia. The characteristic x-ray findings were generalized decalcification, bone tumors, multiple bone cysts and fish-type vertebral bodies. However, the earliest presenting symptom may be in the mouth, as a considerable number of the cases reported in the present paper indicate.

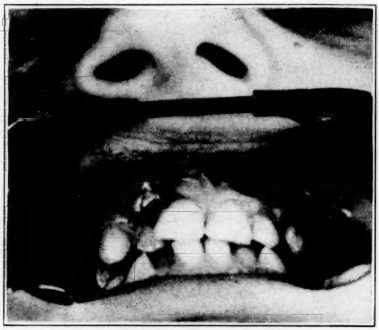

FIGURE 3. *Visible and Palpable Tumor.*

This growth was biopsied and proved to be a giant-cell tumor. Note the cyanotic enlargement over the right lateral incisor and the partial obliteration of the labial fold. The patient was a young girl.

Tumors

Of the 45 cases, about half showed tumors of the jaw (Figs. 3 and 4); one presented large, smooth masses in both maxillary cuspid regions that were difficult to differentiate from the swellings normally found prior to the eruption of the maxillary cuspid; several presented soft-tissue masses that might have been taken for ordinary benign giant-cell tumors.

In view of the similarity in histologic structure between the benign giant-cell tumors often found in the mouth and the giant-cell tumors found in osteitis fibrosa cystica, every case presenting a nonmalignant tumor of the soft tissue should be investigated for the possibility of hyperparathyroidism. This also applies to certain types of bony growths, which will be spoken of below.

Malocclusion

In several cases, malocclusion or jaw distortion was one of the earliest presenting symptoms (Figs. 1 and 5). Prognathism of the mandible, which develops suddenly and without any apparent cause, especially in adults, should be regarded with suspicion. If such prognathism is associated with separation of the teeth, it may be an important clue to the early recognition of the disease. Likewise, the sudden separation of the teeth in any locality of the maxillas or mandible is to be regarded with suspicion, since it may be evidence of a tumor that by its proliferation has mechanically caused a separation of the teeth. Certainly such conditions warrant thorough x-ray study. Figure 1 shows the protrusion of the lower jaw in a patient who previous to the onset of the ailment had normal occlusion.

Cysts

Perhaps the most striking change in the jaw bone is the occurrence of cystlike cavities visible by x-ray (Figs. 2 and 6). These may be unilocular or multilocular and may or may not be associated with pulp disease. In advanced cases the entire mandible may be honeycombed by multiple cystic cavities. Cysts of unknown origin that seem to occur independent of apical disease should create in the observer a desire to know more about their etiology.

FIGURE 4. *Distortion of the Upper Right Maxilla Due to Hyperparathyroidism.*

Caries

Paradoxical as it may first seem, caries is diminished (Figs. 2 and 7). This finding at first caused great interest. Here was a disease in which calcium was being extracted from every

bone in the body and was being excreted. If it were possible to extract calcium from already formed teeth in this disease, one would expect

FIGURE 5. *Disturbance of the Occlusion and Movement of the Teeth.*

Note the space that has occurred between the upper central incisors and between the upper right lateral and central incisors. These were not present one year previously. No tumor is visible or demonstrable, but the general decalcification of the jaws has permitted the forces of mastication to move the teeth in the direction of least resistance.

widespread caries. Instead of this, one finds the opposite. This phenomenon, which is consistently observed, indicates that calcium cannot be withdrawn through the blood stream from already cal-

FIGURE 6. *Changes Due to Hyperparathyroidism.*

Note the mandible honeycombed by multiple cystlike cavities, which may resemble the cysts that result from periapical disease.

cified dental tissues. This opinion has found general recognition in the literature.

Osteoporosis

In the x-ray films of patients with bony changes due to hyperparathyroidism, the teeth stand out so that they seem in contrast radiopaque, whereas the bone is radiolucent (Figs. 2 and 7). In other words, in osteoporosis of the supporting bone, when calcium has been withdrawn, the teeth remain at their usual radiopaque level.

Bone Trabeculae

What bone remains has very finely meshed trabeculae, which can hardly be seen, and appears in a symmetrical pattern of lacelike delicacy, often described by radiologists as resembling ground glass (Figs. 2 and 7).

Lamina Dura

The most striking finding is the one for which every dentist should be on the watch, since it must suggest hyperparathyroidism although it is also seen in diseases such as osteomalacia.[10] It is the absence, or partial loss, of the lamina dura — the thin layer of compact bone that abuts the periodontal membrane (Figs. 2 and 7). This structure

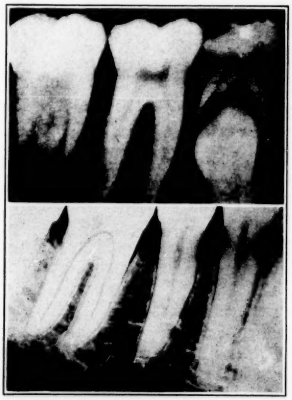

FIGURE 7. *Changes Due to Hyperparathyroidism.*

In the upper film, note the lack of lamina dura, the ground-glass appearance of the bone (closely meshed trabeculae) and the well-formed teeth, compared in the lower film with a normal lamina dura and normal trabeculation.

has been called the periodontal lamella, the lamina dura and the linea alba. In the x-ray films, the normal image of the lamina dura is a fine, even white line less than 1.0 mm. wide, with a slight thickening at the cementoenamel junction on each side. It is never any wider except in early life or because of disease. The lamina dura is perhaps the most delicate layer of compact bone in the body, and it is the first bony barrier that infection from caries in a tooth meets in its effort to extend itself into the tissues of the body.

The lamina dura may be entirely absent, or it may be deficient in only a few localized spots in osteitis fibrosa cystica. In the less marked cases it may be merely extremely thin.

Summary and Conclusions

The frequency with which oral symptoms appear in hyperparathyroidism suggests that manifestations about the teeth are of value in recognition of the disease.

The dental symptoms are visible or palpable tumors of the jaw, malocclusion or distortion of the normal arrangement of the teeth (motility), cystlike cavities of the jaws, diminished dental caries, osteoporosis, closely meshed trabeculae and absence of the lamina dura.

The fact that, even in extreme cases of demineralization in hyperparathyroidism, caries does not increase is convincing evidence that the resorption of calcium and phosphorus from mature teeth is impossible by way of the blood stream.

Disorders of the parathyroid gland should be further studied to throw light on the etiology of dental diseases.[11]

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MEDICAL PROGRESS

PATHOLOGY

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BOSTON

THE experimental production of cirrhosis of the liver and its relation to the disease in man was thoroughly reviewed by Moon[1] in 1934. A brief summary will serve as a background for the interesting developments in the intervening years. Agreement was quickly reached by all experimenters that a biliary type of cirrhosis essentially identical with the human lesion could be produced by ligation of the common bile duct. Cirrhosis of the portal or Laënnec type was first produced by Wegner[2] with phosphorus in 1872. In subsequent years, success was claimed for many other methods, all dependent on the use of one or another hepatotoxic agent. The methods can roughly be divided into two categories: the prolonged administration of minute, individually nontoxic doses of agents such as phosphorus,[2] arsenic,[3, 4] lead,[5, 6] copper,[7] manganese,[8, 9] silica[10] and tar[11]; and the repeated use of toxic but nonlethal doses of powerful organic liver poisons, particularly halogen compounds such as chloroform[12, 13] and carbon tetrachloride.[14, 15] Opie[16] added another factor by combining toxic doses of chloroform with the injection of colon bacilli intravenously, but it is improbable that the experimental production of cirrhosis has ever been accomplished by the use of infectious agents alone.

The cirrhoses produced resembled in varying degrees atrophic cirrhosis as it is seen in man. Using copper, Mallory, Parker and Nye[7] produced a cirrhosis with pigmentation reminiscent of hemochromatosis. Phosphorus and lead administration were shown by Mallory[6, 17] to result in cirrhosis, with hyaline degeneration of the liver cells similar to that seen in alcoholic cirrhosis of man. Other characteristic features of the human lesion, such as extensive fatty vacuolization and reconstruction of the liver architecture, were not observed. The cirrhosis produced with carbon tetrachloride and related compounds exhibited, in the most successful experiments, pictures closely resembling the so-called "toxic" or postatrophy cirrhosis of human beings. Throughout the long list of demonstrated effective agents, not one can be singled out that seems a probable cause of a significant proportion of cirrhosis in man. Most efforts to induce cirrhosis with the agent that clinical experience rates most important — ethyl alcohol — were flatly unsuccessful, and the few re-

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ported positive results were at best equivocal. After critical survey, Moon, in agreement with the opinion of the vast majority of pathologists, decided that no credible evidence of the experimental production of cirrhosis with alcohol existed.

That alcohol was not without effect on the liver could not, however, be denied. Bollman and Mann,[15] like others before them, produced large fatty livers by prolonged administration of alcohol to dogs, but necrosis and fibrosis did not occur. Moreover, the administration of alcohol to dogs subsisting on a high-fat diet, which, it is well known, will of itself produce a fatty liver, markedly accelerated the rate of fat infiltration, but again no cirrhosis resulted. An adjuvant effect of alcohol in increasing the toxicity of various agents on the liver was reported by Fischler[18] following the use of phosphorus, and by Lamson and Wing[14] with carbon tetrachloride. Bollman and Mann[15] have confirmed both experiments.

Despite the failure of so many experienced investigators to demonstrate the progression of a chronic fatty infiltration into a cirrhosis, numerous observations during the last two years indicate that this frequently occurs if the experiment is sufficiently prolonged. Allan, Bowie, McLeod and Robinson[19] noted that in depancreatized dogs kept alive with insulin for long periods fatty livers regularly develop. Chaikoff, Connor and Biskind[20] found that in similar animals surviving for periods of two to four years the fat gradually disappeared from the liver, which grew progressively smaller and in many cases became definitely cirrhotic. The resulting cirrhosis closely resembled so-called "alcoholic cirrhosis" of man. The parallel to the not infrequent large fatty livers and the occasional development of cirrhosis in human diabetic patients was pointed out.

Pursuing this clue with vigor, Connor and Chaikoff have reported[21] the development of cirrhosis in 4 of 16 dogs receiving a high-fat diet in addition to large doses of alcohol, and even in a number of dogs treated with a high-fat diet alone.[22] It is interesting that in the latter the scarring was more diffuse and less similar to human portal cirrhosis than in the dogs that had received alcohol as well. Similarly, in rabbits, which were fed a low-carbohydrate, high-fat diet and given alcohol by stomach tube for weeks and months to the limits of tolerance, Connor[23] reports the appearance of cirrhosis in 7 of 20 animals surviving the regime for more than a few weeks. In many of the degenerating cells, coarse "hyaline" networks developed in the cytoplasm of the hepatic cells, corresponding to the picture regarded by Mallory as pathognomonic for alcoholic cirrhosis. In human alcoholic patients Connor[24] has also been able at the autopsy table to trace all stages of progression from the large smooth fatty liver, which is the most constant anatomic finding in chronic alcoholism (Fahr,[25] LeCount and Singer[26]), through a hypertrophic stage of cirrhosis to eventual atrophy, a progression fully confirmed in a similar study by Hall and Morgan.[27]

Connor's success when so many others have failed can scarcely be attributed to persistence alone, and constitutes a challenge to further study and experiment. Connor himself argues for a direct metabolic effect of alcohol and suggests that it interferes with normal tissue oxidative mechanisms, as do many of the other hepatotoxic agents, such as phosphorus and carbon tetrachloride, and as certainly happens in uncontrolled diabetes. Direct evidence that ethyl alcohol exerts such an effect on the liver appears to have been furnished by Newman, Van Winkle, Kennedy and Morton.[28] Several other possibilities, however, are suggested by recent investigations.

Among the multitude of toxic effects attributed to alcohol in the past, recent investigation has proved one after another — peripheral neuritis[29] and chronic myocarditis,[30] for example — to be in fact manifestations of vitamin deficiency curable in many cases without deprivation of alcohol by the administration of vitamin concentrates. In this respect, it is interesting that Rich and Hamilton[31] have been able to produce cirrhosis in rabbits with a diet deficient in vitamin B. The exact nature of the deficiency is still undetermined. Evidence points to a factor present in yeast other than vitamins B_1, B_2 and B_6 or nicotinic acid. In experiments carried from twenty-five to one hundred and thirteen days, the livers were smooth to finely granular, cut with increased resistance, and showed a diffuse cirrhosis, which was at first limited to the portal areas but later invaded the lobules. Fat was present in some specimens, absent in others. Necrotic liver cells were usually difficult to find. The cirrhosis evidently closely resembles and may actually prove to be indistinguishable from alcoholic cirrhosis in man.

Significantly different effects were obtained by György and Goldblatt,[32] who used another experimental animal, the rat, with a similar deficiency of an unidentified radicle of the vitamin B complex. In contrast to the rabbits of Rich and Hamilton, the rats developed extensive focal necroses with precirrhotic changes resembling the picture of acute yellow atrophy in man. The addition of yeast extract regularly prevented this hepatic injury. Further evidence of an important protective factor in yeast are the reports by Von Glahn

and Flinn[33] that the addition of brewer's yeast to the diet diminishes or prevents damage due to arsenic, and the observations of Rhoads[34] of a similar effect on poisoning with "butter yellow."

Still another possible mechanism of liver damage with resultant cirrhosis has been brought to light by recent studies of the metabolism of choline and of certain amino-acids, in particular cystine and methionine. Diets deficient in choline have been shown by Best et al.[35] to produce large fatty livers, and conversely the administration of choline will rapidly reduce the fat deposits in the livers of depancreatized dogs and protect against the effects of high-fat diets and of alcohol. Although the production of cirrhosis by choline deficiency alone has yet to be reported, it seems highly probable that any method which will produce a chronic fat infiltration of the liver will, if pursued with sufficient tenacity, result eventually in cirrhosis.

It has been known for a long time that cystine exerts a toxic effect on the kidneys, producing extensive hemorrhagic necrosis. Less notice has been given to hepatic degeneration, although numerous investigators have observed it. Earle and Victor[36] report a series of experiments in rats in which for reasons as yet undetermined little nephrotoxic effect was observed with additions of 5 and 10 per cent of cystine to the diet, but extensive hepatic injury and true cirrhosis developed. Important in this regard is the demonstration by Griffith and Wade[37] that the effects of choline and cystine are antagonistic in the production of fatty livers. The addition of cystine to the diet increases the requirements for choline. Methionine has an opposite effect and tends to protect the liver from the results of choline deficiency.

Finally, one must take cognizance of the increasing evidence that protein depletion, as well as that of carbohydrate, may markedly enhance the susceptibility of the liver to various toxic agents; in fact, Ravdin et al.[38] have shown in experiments with chloroform that the necrosis is independent of the glycogen content of the liver cells, and Von Glahn and Flinn[33] have made the same observation in experiments with arsenic. Miller and Whipple[39] and Ravdin et al.[40] agree that protein depletion significantly increases the susceptibility of the liver to chloroform and also diminishes its regenerative capacity. Further confirmatory evidence of the importance of adequate protein intake is provided by the observations of Smith[41] in regard to the cirrhosis produced by selenium and of Messinger and Hawkins[42] in that caused by arsphenamine.

Brief though this review has been, the complexity of the field must have been made apparent. Cirrhosis of the liver of the portal or Laënnec type is unquestionably the end result of chronic or repetitive hepatic degeneration, and hence its etiology is that of the underlying injury of liver cells. The long list of hepatotoxic agents has again been extended, but still fails to include, except for the controversial ethyl alcohol, any substance that seems a probable cause of cirrhosis in man. Far more important has been the demonstration that dietary and metabolic factors can produce hepatic degeneration. As Best et al.[35] remarked in 1934, "Dietary methods are now available both for producing fatty livers and for preventing them by the use of substances, sterols, fats and phosphatids, which are normal constituents of both plant and animal cells." It seems probable today that the statement could be made with equal accuracy of cirrhosis. Imbalances of metabolism or of dietary intake and specific nutritional deficiencies can produce hepatic degeneration and hence cirrhosis. Such factors can enormously enhance the toxicity of many agents in their effect on the liver cell, and by the unbalancing of antagonistic factors a group of individually ineffective influences may add up to a total that is capable of producing irreparable liver damage. Whether so comparatively innocuous a substance as ethyl alcohol can become an effective liver toxin under such conditions or whether alcohol maintains its status as the most important cause of cirrhosis in man simply because it is the most frequent cause of a certain type of nutritional deficiency is still unanswerable. The solution to this rather academic question demands still more patient experimentation under most rigidly controlled conditions.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

CASE 27241

PRESENTATION OF CASE

First Admission. A fifty-six-year-old woman entered the hospital complaining of recurrent attacks of severe crampy abdominal pain of approximately two years' duration. The pain tended to be on the right side of the abdomen, often radiated to the right flank and costovertebral angle, and lasted about two hours. This symptom was accompanied by urgency, frequency, burning and nocturia. X-ray examination showed a moderate dilatation of the right ureter without apparent cause, and kidney outlines and function tests were normal. Cystoscopic examination revealed an edematous right ureteral orifice, with surrounding cystitis cystica.

The patient returned five months later with the same complaints, and tubercle bacilli were found to be coming from the right kidney. A right nephrectomy was performed, and pathological study confirmed the diagnosis of tuberculosis. During hospitalization, a gastrointestinal series, barium enema and Graham test were negative.

Final Admission (four years later). In the interval the patient had felt perfectly well until two months before admission, when she began to suffer from attacks of heavy, sometimes crampy nonradiating pain in the right side of her abdomen. The pain was not too severe, although it sometimes precluded sleep; it was more intense after meals, but it was apparently not governed by the type of food eaten. The attacks occurred at intervals, although never separated by more than a week. There were accompanying anorexia and a weight loss of ten pounds. The patient denied urinary symptoms or change in bowel habits or the character of her stools.

The patient had had measles, scarlet fever, typhoid and pleurisy. Twenty years before admission, a panhysterectomy and appendectomy had been performed, and some time later a low back fusion for tuberculosis of the spine. X-ray films showed fusion of the vertebrae from the eleventh dorsal to the third lumbar.

The patient's mother had died at fifty-four of typhoid fever, her father at sixty-eight of a stroke. Several members of her mother's family had died of tuberculosis.

On examination the patient looked tired and showed evidence of recent weight loss, but she was not acutely ill. The heart and lungs were essentially normal, the blood pressure 105 systolic, 60 diastolic. The abdomen was slightly distended, and a moderately tender, hard, somewhat irregular mass could be felt both front and back filling the right side. It moved slightly with respiration, appeared to be below the liver area, and apparently was not connected with it. Extension of the thigh with the patient lying on her stomach produced no pain. A pelvic examination was negative.

The temperature was 99°F., the pulse 88, and the respirations 20.

The urine showed a + test for albumin, and the sediment contained ten white blood cells and an occasional red blood cell per high-power field. The blood showed a red-cell count of 4,500,000 with a hemoglobin of 11.3 gm. (photoelectric-cell technic), and a white-cell count of 15,200 with 93 per cent polymorphonuclears. The serum van den Bergh was normal, and the blood Hinton reaction negative. The stools were normal.

A plain x-ray film of the abdomen showed a large, sharply defined mass in the right midabdomen, which was distinct from the lower border of the liver and measured 12 cm. in diameter. A moderate quantity of gas was present in the colon, but no dilated loops of intestine were seen.

A barium enema met an almost complete obstruction in the right side of the transverse colon. A large area of ulceration was present, with marked narrowing in the proximal end of the lesion, the area corresponding to the large palpable mass. The cecum was not dilated, and only a small quantity of gas was seen in the small bowel, without dilatation.

A Graham test was positive. There was a spinal fusion of the upper lumbar vertebrae; the bones of the pelvis and dorsal spine appeared normal.

X-ray study of the chest showed marked prominence of the heart shadow in the region of the left ventricle. The lung fields were clear.

An operation was performed on the eighth hospital day.

DIFFERENTIAL DIAGNOSIS

DR. RICHARD H. SWEET: By the efforts of the surgeon, we can exclude several organs from the differential diagnosis.

The stools were normal. I cannot refrain from saying that that is a significant statement if true, because as I reflect on this case the differential diagnosis can be brought down to deciding between two or three very definite conditions, all of which usually cause bleeding into the stools. Yet

the record says the stools were normal. I hardly believe it, but we shall have to accept that statement in discussing the differential diagnosis.

DR. TRACY B. MALLORY: Only one stool examination was performed, and it was negative.

DR. SWEET: It is not very unusual to have what appears to be a complete obstruction by barium enema without clinical evidence of obstruction, such as distention. The barium enema may fail to pass even when intestinal contents may pass through from above. Is that not so, Dr. Holmes?

DR. GEORGE W. HOLMES: Yes, that is true.

DR. EDWARD F. BLAND: May I interpose here? I happen to have seen the patient. The record does not mention that a Miller–Abbott tube was passed shortly after.

DR. SWEET: That might have accounted for the absence of gas. May we see the x-ray films?

DR. HOLMES: The problem, of course, is to decide what the obstruction was due to. That there was obstruction is quite obvious. It is a well-known fact that when one gives a barium enema, as Dr. Sweet pointed out, the amount of obstruction always appears greater than it does when the barium is given by mouth. That is particularly true if the lesion in the bowel is fungating. It may form a cone so that the contents of the bowel pass through readily from above, but when the flow is started in the other direction there is a valve effect. I do not know how much one can rely on it, but it is of some value. It suggests that the growth is into the lumen of the bowel, not a simple ulceration. The Miller–Abbott tube is seen in position there. There is rather more gas in the bowel than the impression gained from the text would indicate. I do not believe that the bowel is definitely dilated, but there is more in the large bowel than one would expect. Here you see the evidence of obstruction, with the colon fairly well filled, and in this film, which was taken after evacuation, the normal mucosal pattern is shown throughout the large bowel, except for the area at the point of obstruction; I should say that there is absence of mucosa into a definite conelike effect, which we usually associate with carcinoma. Apparently, the lesion is single. The remainder of the bowel is extremely normal for tuberculosis. This film shows the meal given by mouth and does not give much added information. Here is a film taken of the chest, and it rules out metastasis to the lung and also evidence of active tuberculosis. As you know, ulcerative lesions in the colon are a little commoner in active tuberculosis, and we have no evidence of that here.

DR. SWEET: The differential diagnosis obviously can be narrowed down very promptly to deciding, as Dr. Holmes has pointed out, between carcinoma and tuberculosis. The catch is that the patient had a lot of tuberculous infection elsewhere, but I presume that she had a carcinoma of the colon. The history would go with either lesion. She had attacks of pain, suggesting obstruction. She had intermittent attacks occurring at frequent intervals, I presume growing severer as time went on. The obstruction in itself might account for a certain amount of weight loss, anorexia and so forth, so that one need not postulate neoplastic disease to explain that symptom necessarily. Let us assume that this is carcinoma of the colon. One or two things about the case are unusual. A carcinoma in the right half of the colon is often associated with a severe degree of anemia. This patient had a red-cell count of 4,500,000, with only a fairly low hemoglobin. Oftentimes the degree of anemia that we see in carcinoma of the cecum and ascending colon is very severe. That, of course, would not exclude it. Again, the stools were normal. I do not believe that any carcinoma of the colon ever existed without microscopic evidence of blood in the stools. On the other hand, the history is very short and is quite consistent with carcinoma. The location in the bowel, that is, not down at the ileocecal region, but farther up in the region of the transverse colon, is a little more in favor of carcinoma than tuberculosis; also, as Dr. Holmes pointed out, in a tuberculous lesion of this type we are accustomed to seeing evidence of disease in the chest, and although that need not necessarily be the case, it seems to me that we should expect it.

On looking at the case from the standpoint of tuberculosis, there are many things in favor of that diagnosis: the facts that the patient has had active tuberculous disease in the spine and in the right kidney, and that she had a positive family history. The clinical history of the case, the intermittent obstruction and so forth in the presence of the palpable mass would go with such a diagnosis perfectly well. But, as I have said, there is absence of active pulmonary disease, and the history is rather short. I should expect also to find a certain amount of blood in the stools with any ulcerative lesion, whether tuberculosis or carcinoma. We might mention one or two things in passing. I thought about actinomycosis until I saw the x-ray film, but now I should exclude it. Usually in association with that we see fistulas, and there were none in this case. Such things as lymphoma might be spoken of. Lymphoma of the colon is not at all unusual. I do not know enough about the interpretation of the x-ray films to know whether it has a characteristic appearance or not.

DR. HOLMES: I do not believe one can distinguish it from a carcinoma, but it is often a mul-

tiple lesion. I did not mention the possibility of a mass outside the colon surrounding it and pressing on it. The size of the mass and the fact that there was no bleeding would lead one to think of such a condition, but I do not believe it could destroy the mucosa to the extent that is present here.

DR. SWEET: Thus the fact that the mucosa had been destroyed, with evidence of ulceration, makes it appear that this was an intrinsic lesion of the bowel wall. I do not think we can go farther in discussing the differential diagnosis. At least, I cannot. Benign tumors, lipoma, myoma and so forth, would not fit the x-ray picture. Oftentimes, in my experience on exploring these doubtful cases, it is difficult by palpation or inspection of the lesion, to distinguish between neoplastic disease and tuberculosis. I think there is a little more evidence in favor of carcinoma than tuberculosis. That is as far as I can go.

DR. JOHN D. STEWART: What significance, if any, do you attach to the positive Graham test?

DR. SWEET: I noted it and meant to speak of it. I have excluded it in my mind because of the physical examination of the tumor. The tumor might have been the gall bladder pressing on the colon, but the remark is made that it was well below the liver edge, and also the x-ray film seems to put it away from the region of the liver. Of course, the kidney can be excluded because it was taken out, as was the appendix; but the patient did have that change from a negative to a positive Graham test, it is true.

CLINICAL DIAGNOSIS

Carcinoma of transverse colon.

DR. SWEET'S DIAGNOSIS

Carcinoma of transverse colon.

ANATOMICAL DIAGNOSES

Carcinoma of transverse colon, with perforation.
Pericolic abscess.
Polyposis coli.
Aspiration pneumonia.
Ileotransverse colostomy.
Operative scars: right nephrectomy; hysterectomy; bilateral salpingectomy, left oöphorectomy, appendectomy.

PATHOLOGICAL DISCUSSION

DR. MALLORY: The patient was explored, and a very large mass was felt in the transverse colon, which was adherent to the liver, omentum and gall bladder. It seemed inoperable and an ileotransverse colostomy was done, short-circuiting the bowel about the lesion. In the course of the operation, difficulty occurred in anesthesia, and a large amount of gastric contents was vomited and aspirated. The patient went promptly into severe shock. She never recovered in spite of two 500-cc. transfusions, and died in a little less than twenty-four hours. At post-mortem examination, the effort was made to free up the mass, and a large abscess cavity was broken into. This showed free communication with the bowel through a perforated cancer of the transverse colon. The peritoneal cavity was quite free from reaction, and the abscess had been very well walled off. The remainder of the autopsy showed extremely wet hemorrhagic lungs, with a great deal of edema and fulminating but incipient pneumonia. There was no evidence at the time of autopsy of any active tuberculous process in the lungs, the urinary tract or the bowel. Five or six polyps were found in the colon, besides the carcinoma.

CASE 27242

PRESENTATION OF CASE

A thirty-nine-year-old woman entered the hospital complaining of blood and pus in her stools.

The patient felt perfectly well until seven weeks before admission, when she first noticed blood in her stools. Several times a day for the next four days when she attempted to have a bowel movement, nothing passed but blood, each time amounting to half a cupful. Then the patient took a laxative, and watery movements colored with blood resulted. Thereupon she consulted her physician, who made a diagnosis of hemorrhoids and prescribed a local ointment and petrolagar. There was no improvement, however, and the loose bloody stools continued. Two weeks before entry the patient consulted another physician, who advised admission to another hospital. Here a hemorrhoidectomy was performed, and the patient stated that "an abscess was found above the hemorrhoids." A rectal tube was used for three days postoperatively, and it drained a purulent material. Thereafter daily enemas were instituted, and the patient found that she was incontinent of feces, which appeared to consist of nothing more than pus. The patient had not suffered from nausea, vomiting or pain, nor had there been weight loss, but on the fourth postoperative day she believed that she had had a chill followed by fever.

On examination the patient was flushed, acutely ill and dehydrated. The heart and lungs were normal, the blood pressure 95 systolic, 60 diastolic. The abdomen was tense, but not tender. No organs were palpable, but one examiner believed a mass might be present in the left lower quadrant.

Around the anus, which drained a foul purulent discharge, were radial scars, and no sphincter

could be felt. The rectal mucosa was redundant, thick and granular, and bled easily. The examiner believed a palpable resistance was present that corresponded to the mass he had felt in the abdomen and that this mass narrowed the lumen of the bowel which was otherwise freely patent.

On vaginal examination the cervix was found to be reddened, slightly patulous and soft; it oozed a purulent discharge, which contained three or four small pieces of red-black friable material. There was slight thickening in both vaults, and the uterus was enlarged to about four times its normal size and was hard and fixed, but not tender.

The temperature was 101°F., the pulse 100, and the respirations 20.

The urine gave a ++ test for bile. The blood showed a red-cell count of 3,410,000 with a hemoglobin of 70 per cent, and a white-cell count of 6600. The nonprotein nitrogen of the blood serum was 40 mg. and the protein 6.8 gm. per 100 cc., and the chlorides were 87.9 milliequivalents per liter. A blood culture and a blood Hinton test were negative. A Frei test was negative, the cervical culture was negative for Beta hemolytic streptococci and a culture of the anal discharge yielded *Pseudomonas aeruginosa*, but no pathogens. A biopsy of the rectal mucosa showed acute and chronic inflammation.

The patient's condition never improved, foul pus continued to drain from the rectum, and on the third hospital day her abdomen was distended, the temperature 103°F. and the white-cell count 8100. A flat abdominal x-ray film showed a large amount of gas in a dilated colon, the greatest dilatation extending from the cecum to the splenic flexure. Some gas was present in the descending colon, however. The presence of air under the diaphragm could not be determined definitely. On the fourth hospital day a cecostomy and appendectomy were performed. The cecum was found to be dilated and so tense that aspiration with a needle attached to a suction tube was necessary before introducing the cecostomy tube. Histologic examination of the appendix showed acute inflammation. Although the patient's abdomen was deflated on the following day and peristalsis was active, her temperature rose to 104°F., and the white-cell count dropped to 3150. Large quantities of foul, bloody material continued to escape from the rectum, with but little discharge from the cecostomy opening. Death occurred on the third postoperative day, seven days after admission.

Differential Diagnosis

Dr. Allen G. Brailey: May we see the x-ray films?

Dr. George W. Holmes: These films confirm the statement made in the text that there was no visible air below the diaphragm on either side and that the colon was enormously dilated. One gets a different impression from looking at the film than from reading the text, since the dilatation is much more than an ordinary one. The haustral markings are visible. It is a queer-looking colon for obstruction. No fluid is present. It seems to stop somewhere in the region of the splenic flexure. The bowel below the splenic flexure does not seem to be dilated although there is some air in it.

Dr. Brailey: In discussing this case, we must answer two questions: First, What was the primary condition from which the patient was suffering, and, secondly, Why did she die? I prefer to answer the second question first. I think she died of overwhelming sepsis, a conclusion that is supported by the record of high fever, falling white-cell count and rapidly fatal course. The urine was said to contain a ++ test for bile, so that I conclude that her liver was seriously damaged, and it may well have contained multiple abscesses, metastatic from the rectal bed. It is not clear whether the patient had generalized peritonitis as well as pelvic peritonitis, which she certainly had. The abdomen was "tense but not tender." Apparently the service suspected intestinal perforation, for an attempt was made to demonstrate air under the diaphragm by x-ray study.

The primary condition was some fulminating, destructive and ulcerating lesion of the rectum. In deciding its nature, I think we should consider carcinoma, ulcerative colitis, lymphogranuloma inguinale, amebiasis, tuberculosis and perhaps some rare infection such as actinomycosis. This was another case of bleeding from the rectum that was not due to "hemorrhoids." In fact, operation in this case may have opened the way for fatal spread of the infection, for the patient appears to have been singularly free from any constitutional symptoms until shortly after the operation was performed.

There is something to be said in favor of carcinoma in this case. Rectal bleeding should always suggest the possibility of carcinoma. A left lower-quadrant mass was felt both through the abdominal wall and by rectum. The uterus is said to have been enlarged, hard and fixed, which might suggest extension of neoplastic disease from the rectum. But I find it impossible to believe that a woman of thirty-nine who had no evidence of ill health eight weeks before death could nevertheless have had cancer so widespread and so ready to break down that it could produce this picture of extensive ulceration and fatal sepsis, all within a relatively few days.

I am even more willing to exclude tuberculosis

of the rectum. It is not a very common disease in this part of the world at present. Its progress is usually slow and chronic, accompanied by the development of multiple fistulas. Certainly tuberculosis alone will not explain this very rapid course. Amebiasis requires a little more attention. It can occur in very acute form, and may be accompanied by the discharge of a great deal of blood and pus. The inflammatory process and even the amebic infection itself may spread to involve adjacent pelvic organs, and, of course, its tendency to produce liver metastases is notorious. Usually this severe form is accompanied by marked pain and tenesmus, but I cannot understand why this woman did not have bowel pain, whatever her diagnosis. But amebic infection is rare in this vicinity, and this is not the common picture of amebiasis, even when it does occur, so that I am inclined to discard amebic infection as a very likely possibility. Incidentally, the disease is rather commoner in the proximal than in the distal end of the large bowel.

I am left with idiopathic ulcerative colitis and lymphogranuloma inguinale. As a matter of fact, this is an excellent description of acute lymphogranuloma involving the rectum. Almost an identical case history is given by Roadaniche, Kirsner and Palmer* in a discussion and comparison of these two diseases. The free discharge of pus and blood is characteristic. A rather serious objection is raised by the report that a Frei test was negative. A Frei test properly performed and with a satisfactory antigen is quite specific. Of course, no laboratory test is infallible, and, in fact, a few very interesting cases have been reported of very ill persons with lymphogranuloma whose Frei tests remained negative until they began to improve. These negative results have been ascribed to a state of anergy. I am therefore not willing to exclude the possibility of lymphogranuloma because of a negative Frei test, but I do think it becomes improbable.

I think that this picture could have been due to fulminating ulcerative colitis. I confess I am puzzled that this patient presented no symptoms whatever except rectal bleeding until about two weeks before her death. I am surprised that she did not have colic, tenesmus and diarrhea, but I think that the lack of these symptoms is hard to explain on any basis. I suppose that the pathologic process must have been relatively mild during the first five or six weeks of her illness, with perhaps only a few ulcerations, which happened to bleed freely, and that shortly after her operation its virulence suddenly mounted and secondary invaders began rapidly to destroy the mucosa and apparently even the anal sphincter. I do not know whether the anal sphincter was actually destroyed by the disease process or whether its function was interfered with in some way. The x-ray films seem more striking than I had thought they would turn out to be from the record, but I do not believe there was mechanical obstruction. I think this was perhaps ileus in a terribly sick woman.

Dr. Richard H. Sweet: That degree of dilatation in the colon is not inconsistent with a fulminating ulcerative colitis.

Clinical Diagnoses

Perianal and pelvic abscess.
Septicemia.

Dr. Brailey's Diagnosis

Acute idiopathic ulcerative colitis.

Anatomical Diagnoses

Acute idiopathic ulcerative colitis, with perforation.
General peritonitis.

Pathological Discussion

Dr. Tracy B. Mallory: At post-mortem examination, a generalized peritonitis was found, with a perforation in the lower sigmoid just barely above the pelvic floor. When the large bowel was opened, it showed multiple areas of erosion, interspersed with polypoid hypertrophied fragments of the remaining mucosa extending all the way from the rectum to the cecum. When one held it up to the light, there were multiple areas where the wall of the bowel was only of paper thickness, and any one of a score of places might easily have perforated. There was no mechanical obstruction.

Nothing was found at autopsy to explain any palpable mass in the left lower quadrant. The uterus and adnexa were normal, and there was no evidence that there had been a localized pelvic abscess before the generalized peritonitis occurred.

Dr. Brailey: What about the liver?

Dr. Mallory: It was negative grossly, and essentially negative histologically.

Dr. Brailey: Why was the patient jaundiced?

Dr. Mallory: It was not apparent that she was.

Dr. Brailey: How could she have a ++ test for bile without being jaundiced?

Dr. Mallory: I do not have any answer to that.

Dr. John D. Stewart: It is a fairly rough test.

Dr. Brailey: Yes; I think that it is.

*Roadaniche, E. C., Kirsner, J. B. and Palmer, W. L. Lymphogranuloma venereum in relation to chronic ulcerative colitis. *J. A. M. A.* 115:515-519, 1940.

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THE BISHOP'S POSTSCRIPT

In March, 1939, the Right Reverend William Lawrence, former bishop of the Protestant Episcopal Diocese of Massachusetts, then nearing his ninetieth year, issued his postscript. Long a tiller of the soil from which are harvested the fruits of men's labors, he prepared a new field for the harvest. Over his own signature and in his own hand, under the title "My Postscript," he penned, not laboriously but with infinite patience, some fifteen hundred letters; these letters went to as many friends of the Massachusetts General Hospital, — "doctors, nurses, workers in all departments of the hospital, as well as patients; old friends of the hospital and new friends, rich and poor," — and the purpose was to unite them in one enterprise: the building of a chapel within the hospital.

This chapel, as the Bishop visualized it and as he then expressed its use, would be not a church or a temple or even a chapel in the ordinary sense, for its purposes would transcend those of such denominational structures. Since the hospital opens its doors to succor those of all faiths, races and traditions, so also should a chapel, itself cloistered within the walls of the hospital, afford to those of all faiths, races and traditions a place of spiritual refreshment.

The enterprise prospered, with its significant appeal, quietly and surely. By the fall of 1939 over fifty thousand dollars had been pledged by more than eight hundred persons — one ward of the hospital sent four dollars and thirty cents, with the signatures of forty-one patients.

Soon thereafter the work was in progress, and in April, 1941, without ceremony, the doors of the chapel were opened that all might see its simple, lofty dignity and the rich coloring of its windows and might appreciate it as a place of peace and sanctuary.

As has been said before, the chapel is not a church, for a church implies religious denominationalism. The inspiration and the work of a Christian ecclesiastic, it is dedicated to the use of all who recognize and seek the help of a power greater than themselves; in a great house of physical healing it is a small chamber for spiritual healing.

Such a chapel, representative of all faiths in general, and of no faith in particular, is fittingly erected in a country that has dedicated itself in principle to freedom of thought and of worship, and to equality of opportunity. It is perhaps also significant that there has been faith to build such a structure for permanent use, in a time when destruction apparently reigns supreme.

HEALTH OF THE NATION AND NATIONAL DEFENSE

No one doubts that the national health is inextricably linked with the national defense. Only a healthy and united nation can hope to prepare and carry forward an adequate military program

under present-day circumstances. Nor is there much doubt — some authorities to the contrary — that a disproportionately large percentage of men called under the Selective Service Act are rejected on grounds of physical or mental defects. On the average, approximately 60 per cent of all men in Massachusetts are now being rejected by the physicians of the local selective-service boards, and, of those passed as fit for full military duty, an additional 18 per cent are turned down by induction-board physicians acting directly under the United States Army. Both boards are guided so far as physical requirements are concerned by official regulations (M. R. 1-9), and it is misleading to say that "the point of view of the army and civilian medical examiners might be expected to vary considerably concerning the acceptability of certain risks."[1] Within reason, the rules and regulations are clear and definitive.

It therefore appears that, at least in Massachusetts, over 70 per cent of all men otherwise available for the Army are being rejected for physical or mental defects. Surgeon General Parran has correctly termed this a "national disgrace." The problem of national health is a challenge to the medical profession. It is idle to argue that the Army regulations are unduly strict; for in modern warfare there is no place save for those who can "take it."

On the other hand, it is comforting to know that the tuberculosis rate (standard x-ray films of the chest are taken of all men inducted in Boston) has dropped to the comparatively low rate of 0.5 per cent,[2] and it is of interest to note that rate of rejection for cardiovascular disease among men already passed by their local boards is almost precisely the same as it was in the last war (0.8 per cent as compared with 1.0 per cent).[3] It is discouraging, however, to find that nearly 5 per cent of all men sent as physically and mentally fit to the Induction Center in Boston were subsequently rejected because of neuropsychiatric abnormalities and that from 5 to 10 per cent were returned to their homes because of insufficient teeth. Detailed figures of rejections by the local-board physicians are not available for the entire induction period, but it is disturbing to note that of the first 3000 men examined nearly 15 per cent were rejected because of dental defects and that a very considerable percentage of all causes of rejection by the local-board physicians might properly be regarded as preventable or remediable.

"After all," says one authority, "what can be done for flat feet, bow legs and perforated ear drums?"[1] The implication seems to be that these causes for rejection could not have been prevented and that the medical profession has done its best, for better or for worse. It is true that little or nothing can be done for a long-standing perforation of an eardrum, — once it has occurred, — but many such chronic disabilities could have been prevented by the medical profession; bowlegs — which, incidentally, are not a cause for rejection — would never have developed under proper child hygiene, and certainly the disability arising from "flat feet" can, with a little judicious advice, be adequately and completely corrected in many cases.

Why "whistle in the dark"? Seventy per cent of the young men in this country are being judged by proper authorities to be physically unfit for "full military service." It is for the medical profession on the one hand and the laity on the other to see to it that this percentage is materially reduced, not only in times of war but also in times of peace.

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1. *Medical News*. Medical Society of the State of New York. January 1, 1941.
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3. Report of the cardiovascular consultant at Camp Devens to the Surgeon General of the United States. Unpublished letter dated April 23, 1918.

MEDICAL EPONYM

Horner's Syndrome

Johann Friedrich Horner (1831–1886), professor at Zurich, published an article "Ueber eine Form von Ptosis [Concerning a Form of Ptosis]" in the *Klinische Monatsblätter für Augenheilkunde* (7: 193–198, 1869). A portion of the translation follows:

> Six weeks after the last pregnancy that occurred a year before I saw her, the patient noticed a gradual drooping of the right upper eyelid . . . the pupil of the right eye was found to be definitely smaller than that of the left, the eyeball very slightly sunken. . . .

While the case was under observation, there developed before our eyes gradually increasing redness and heat of the right half of the face, although the left half remained pale and cold. . . . The patient then told us for the first time that she had never perspired on the right side. . . . I believe that in view of all these symptoms no one will question my opinion that this gradually developing, but never complete, ptosis should be regarded as a paralysis of the superior palpebral muscle, which is supplied by the sympathetic. I thus regard the phenomenon in the upper lid as a part of a larger symptom-complex.

R. W. B.

MASSACHUSETTS MEDICAL SOCIETY

SECTION OF OBSTETRICS AND GYNECOLOGY*

RAYMOND S. TITUS, M.D., *Secretary*
330 Dartmouth Street
Boston

HEMORRHAGE AND SHOCK ASSOCIATED WITH CESAREAN SECTION AND FOLLOWED BY DEATH

A forty-year-old para II was first seen in the thirty-ninth week of her pregnancy by the physician who delivered her. She had moved into his neighborhood recently, and previously had presumably received adequate prenatal care. Her first question was, "Will you deliver me by cesarean section? I have such a horror of my first delivery that I just can't bear the thought of going through labor again."

The past history was irrelevant. The first pregnancy two years previously had terminated in a forceps delivery of a living child.

The patient was seen only once before delivery. At that time, physical examination disclosed a well-developed and well-nourished woman. The heart was not enlarged; there were no murmurs. The lungs were clear and resonant; there were no rales. The blood pressure was 120 systolic, 65 diastolic. The abdomen was enlarged to a size consistent with her dates; the fetal heart was audible.

An elective classic cesarean section was performed before labor started, nitrous oxide, oxygen and ether being used as the anesthetic. Two hours after delivery, the patient died from hemorrhage and shock. The abdominal dressing was soaked with blood, and there was reported to have been a good deal of vaginal bleeding.

Comment. One does not expect to see fatal post-partum hemorrhage in elective cesarean section, and in well-trained hands death from this cause two hours after delivery is inexcusable. The main point to be brought out discussing this case is that cesarean section still carries hazards. It is not an operation to be undertaken without appreciating these hazards, and to do cesarean section because a patient states that she cannot stand the pangs of labor is absolutely unwarranted in this day of comparative freedom from pain afforded by modern analgesia.

*A series of selected case histories by members of the section will be published weekly. Comments and questions by subscribers are solicited and will be discussed by members of the section.

WHAT A HEALTH EXAMINATION MEANS†

A hundred years ago, when Mr. Jones called in the doctor we knew that he must be very sick, perhaps dying. In those days people did not consult the doctor until they were in real trouble. Yet almost one hundred years ago, in 1850, to be exact, a prominent Bostonian, Lemuel Shattuck, was advocating a measure that called for visits with the doctor when one was not sick at all. At that time, typhoid fever, malaria, smallpox, diphtheria and similar plagues were rampant. The important advances to be made were clearly in the field of public health. Mr. Shattuck believed, nevertheless, that more attention should be paid to personal health; that people should be encouraged to have frequent "sanitary examinations" made of themselves in an effort to promote their health and to prevent disease.

Since that time, there has been a growth of the idea in certain directions, yet there seems to be a lack of enthusiasm for it on the part of the people in general and to some degree within the medical profession itself — the laymen, perhaps because it seems unnecessary to go to the doctor when one is perfectly well; some doctors, because they may think that there is not enough to be accomplished by such measures. Modern medicine surely should not be, as it has been in the past, primarily engaged with death, pain and disease, but should concern itself more with health, happiness and life.

Legend has it that the ancient Chinese paid the doctor so long as the patient remained well. Yet experience shows us that although many Orientals may live to a ripe old age, they all seem to reach the same eventual state of physical dissolution, whether they have paid the doctor to keep them well or not. Now the question is, it seems to me, whether or not such a thing as a periodic health examination can be of any real value in helping us to lead healthier, happier and longer lives.

As a matter of fact, infants and young children have examinations at fairly frequent intervals. Children of school age in many parts of this country, through the agency of the school physician and school nurse, are given some kind of a health check-up at intervals. Many adults undergo an examination when they apply for life insurance. In industry, clinics have been set up for examination of employees. There remains a tremendous group of people, nevertheless, not reached through these channels, people who have no family physician and who lack the initiative to seek out a physician in whom they can place their confidence and to whom they can turn for medical advice.

It seems to me that every adult ought to have a doctor who knows him as an individual person and who has more information about him than mere data regarding his age, weight, blood pressure, and so forth. We are all

†A "Green Lights to Health" broadcast given through Station WAAB by the late Dr. Michael E. Murray, Jr., on Tuesday, December 17, 1940, and sponsored by the Public Education Committee of the Massachusetts Medical Society and the Massachusetts Department of Public Health.

going to need the doctor some time, and calling on a physician who has known us and who has examined us in the past is much more satisfactory from the point of view of both patient and physician than a situation in which an acutely sick person has a strange physician see him for the first time.

Another obvious consideration, of course, in having such a relation with a personal or family physician is that he gives us a thorough examination at the beginning of the relation and re-examines us from time to time for the purpose of discovering physical defects or the signs of early disease, and in general helping us in any way that he can to maintain a state of good health.

It might be well to discuss briefly what such an examination entails. The physician will ask something of one's family history, inquire into the details of one's past illnesses and will also determine whether or not one is having any difficulties at the time of the visit. He will then proceed to make a careful examination, which will include the skin, eyes, ears, nose and throat, heart, lungs, abdomen, the determination of blood pressure, a search for any unusual lumps and so forth. The doctor will also take a urine sample for analysis, and may do a blood test.

My conviction is that too much stress cannot be laid on the value of the history. In recounting the illnesses, accidents, operations and trials of our past lives, we may very well give information of great importance, although it may not appear to us to be of any particular significance. In this connection, I think it is well to remember that the things we tell the doctor are held in confidence by him and there is nothing to be gained by concealing something that may be difficult to talk about. Frequently a good talk with the doctor will help straighten out troubles that are rooted in worry, fear or discouragement. Emotional difficulties, although they may not kill us, can without any doubt make us just as sick and ineffective as the grossest kind of organic disease. An organized set of emotional difficulties, by the way, has a name: it is called a neurosis. There are times when the wise family doctor can do much to help us maintain a solid emotional balance.

To get back to our examination, after the history is taken, a careful physical examination is made, including the procedures already mentioned. I also believe that a Wassermann test or one of its modifications should be done. Further blood examinations may not always be necessary, but a hemoglobin determination and an examination of a blood smear ought to be included in most cases. The white-cell and a red-cell count should be determined, if indicated. In the event that these are all normal, they serve as a base line for comparison if any of them change later in life.

After these things have been done, the doctor will discuss the results of the examination with the patient and will answer his questions as adequately as possible.

Most of us will be found to be in fundamentally sound health or to have only some minor deviation from the normal. We then have the assurance that within the limits of such an examination we are in a state of good physical health.

Sometimes, of course, the physician will find the existence of definite disease and will recommend treatment, or he may find some suspicious signs for which he will ask us to report later for further observation or recommend further immediate investigation, such as chest, stomach or intestinal x-ray study, blood chemistry determinations or an electrocardiogram, these things being done, of course, only when indicated. A metabolism test may in some cases be recommended.

There is no reason to believe that our family doctors are unable or unwilling to take the time and pains to do a good job. Some of us may still have a picture of the family doctor as a kindly old gentleman with long whiskers who hitches Dobbin to the gatepost, comes in for a visit and limits his examination to a look at the tongue and a pat on the back. I suppose such ideas make some people prefer to place themselves in the hands of a so-called "health institute."

I know that we can find plenty of well-trained physicians who are going to give us careful and competent attention, men in whom we can place our confidence. I believe that every adult ought to have such a physician as his own doctor, and that a careful examination should be done at the beginning of this relation. If the person subsequently becomes ill or has some symptoms that worry him, he should report to this same physician. If he remains in a state of good health, he ought to continue to see his physician from time to time — probably best, every year, but certainly, every few years. I prefer to see my doctor every year.

If expert advice or some of the more complicated laboratory procedures are needed, our physician can help us in securing the services of the appropriate specialist. He can also be of tremendous help to the specialist in furnishing background information so necessary for the intelligent evaluation of our particular problem. In this way we can be sure that, so far as the present state of the art and science of medicine goes, we are getting the best. If we happen to be somebody who has a limited income, we can be thankful that we live in this country where through one channel or another good medical care can be had by all. But we have to go and get it. It will not just come to us.

One may ask what kind of abnormality might be discovered at such an examination. Quite a few things might be found, although if many of them existed to any serious degree, the patient would probably have some kind of symptoms. One of the various anemias might be found. Sometimes the finding of a definite degree of anemia leads to the discovery of some underlying cause. Tuberculosis in its earliest stages is usually not detectable by physical examination of the chest, and would not be discovered unless it were sufficiently advanced that the patient had some symptoms or some definite physical signs. Any suspicious symptoms, such as persistent cough or the inability to maintain normal weight, would lead the examining physician to recommend an x-ray examination of the chest for diagnosis. Certain types of early cancer might also be discovered. When such a discovery is made, appropriate treatment can be given with a good chance for complete cure, whereas cancer in an advanced state, as we know, is not nearly so amenable to treatment.

I recently heard of a woman in her forties who, in the course of a routine physical examination, was found to have a small ulcerated area. It was smaller than a dime. A small specimen was taken from the region and was found to be suspicious of early cancer. She is having well-planned treatment, and her chances of getting complete relief are almost 100 per cent. A number of other conditions that might be uncovered are high blood pressure, certain types of heart disease, chronic Bright's disease and diabetes. Complete cures for some of these maladies are not known, but in some conditions, such

as diabetes, the physician can prescribe treatment that will make a long and useful life possible when neglect of the disease would be disastrous. In certain other diseases, modification of one's mode of life, especially with regard to work, diet, exercise and rest, may be of the utmost importance.

One drawback might, I think, be mentioned. An examination that reveals no evidence of ill health may sometimes create a false sense of security. It must be remembered that the physician is not an omniscient being and that some diseases have an insidious beginning, sometimes existing in an early stage without presenting symptoms or signs, and not being apparent even after the most painstaking examination.

Why, then, do we advise these examinations? I think that a question of this nature can be answered best by summing up the points that I have previously tried to make. First of all, I think that we all ought to have a complete examination early in adult life to determine whether or not we have any physical defects or whether we have the early signs of disease. We ought to have such basic information so that we can correct any defects that are amenable to treatment. If we are found to have some condition for which there is no complete cure, we can then, at least, have competent advice in the regulation of our daily lives. Secondly, we should, each one of us, establish a relation with a doctor whom we choose as our physician and medical adviser and whom we can consult subsequently either because of illness or for advice about many of the knotty problems that are bound to vex us from time to time. It is far better to turn to an intelligent physician who has some common sense and both feet on the ground than to take our medical advice from well-meaning neighbors, faddists and cultists.

In conclusion, may I re-emphasize my belief in the importance of having a personal or family physician? Let him give you a thorough examination. Let him see you from time to time. Once a year is not too often. Get your advice about health problems from him. Let him, in what ways he can, help you to keep well, and in time of illness, call on him.

DEATHS

GILMAN — BERNARD B. GILMAN, M.D., of Boston, died May 31. He was in his thirty-eighth year.

He attended Harvard College and received his degree from Harvard Medical School in 1929. Dr. Gilman was assistant director of the division of communicable diseases of the Massachusetts Department of Public Health. In 1938 he received the degree of master of public health at Harvard.

He was a member of the Massachusetts Medical Society and the American Medical Association.

His widow, two sisters and five brothers survive him.

HAIGIS — PETER J. HAIGIS, M.D., of Foxboro, died June 2. He was in his sixty-ninth year. Dr. Haigis received his degree from Boston University School of Medicine in 1904. He was a member of the Massachusetts Medical Society and the American Medical Association.

CORRESPONDENCE

DEPRIVATION OF LICENSE

To the Editor: The license of Dr. James B. Ryan, 53 Maple Street, Easthampton, Massachusetts, was revoked by the Board of Registration in Medicine on May 29 because of deceit in the writing of prescriptions for narcotic drugs.

STEPHEN RUSHMORE, M.D., *Secretary*
Board of Registration in Medicine

State House
Boston

REPORTS OF MEETINGS

HARVARD MEDICAL SOCIETY

There was a regular meeting of the Harvard Medical Society on March 11, with Dr. Elliott C. Cutler presiding. The first case was that of a forty-nine-year-old man who had had, over a period of seven weeks, a gradual onset of painless jaundice, with anorexia but no nausea or vomiting. There had been fleeting right upper-quadrant pain on one occasion three weeks previously. The stools had been light in color, and there had been periodic dark urine. Physical examination revealed marked jaundice, weight loss and an enlarged nontender liver. The chief positive laboratory findings were an elevated icteric index and cholesterol, and clay-colored stools, which were strongly guaiac positive. The prothrombin time was increased, and a gastrointestinal series revealed a filling defect of the duodenum. The patient was started on a regimen of parenteral glucose and vitamin K, but developed a sudden fever on the second day, with a positive pneumococcus blood culture. Treatment with sulfathiazole reduced the fever and the number of colonies on blood culture, but there was still evidence of a bleeding tendency, and blood transfusion and vitamin K were undertaken to return the red-cell count and prothrombin level to normal. Dr. Soma Weiss suggested that the case was probably one of primary neoplasm of the head of the pancreas. Dr. Samuel A. Levine commented that the early detection and intensive treatment of pneumonia may prevent the onset of the characteristic pulmonic manifestations of the disease.

The second case was that of a seventy-two-year-old man who for two or three years had experienced indigestion, gaseous eructations and an idiosyncrasy to fried and fatty foods, all of which were relieved by a proper diet. For the previous six months, there had been an exaggeration of all symptoms, with severe right upper-quadrant pain, nausea and vomiting. Physical examination revealed a dehydrated, questionably jaundiced elderly man, with a large tender right upper-quadrant mass, a leukocytosis of 21,000, an icteric index of 18, a negative diastase test and a slightly increased prothrombin time. On conservative treatment, the abdominal mass diminished in size. A gastrointestinal series was essentially negative, a cholecystogram revealed a nonfunctioning organ, and there was bile in the urine, an increased urobilinogen and occult blood in the stools. The patient was given 10 mg. of vitamin K preoperatively, and when cholecystectomy was carried out, small stones were found in all the ducts.

The speaker of the evening was Dr. Israel S. Ravdin, who discussed "Some Aspects of the Physiology of the Liver and Biliary Passages." Dr. Ravdin compared the liver function to that of a food commissariat. For carbohydrates this organ is the place of synthesis and storage of glycogen, which is the sole source of metabolized glucose. Most of the liver-function tests based on carbohydrate metabolism have been found unsatisfactory either because they remain normal until there is a large percentage of damage (as in the glucose-tolerance test) or because they add nothing to the clinical diagnosis (as

in the galactose test). The protein functions of deamination and the synthesis of serum protein, fibrinogen and prothrombin are now assuming greater importance. There can be as high as 90 per cent destruction of liver tissue, with maintenance of normal deamination. The liver is the largest store of labile protein, and probably the entire source of plasma protein. A decrease in the body protein is evidence of hepatic insufficiency, except in the presence of anemia and nephritis. The liver is the sole source of fibrinogen, which is decreased only in late toxic processes. For the synthesis of prothrombin, the presence of bile salts in the gastrointestinal tract is necessary for the absorption of vitamin K.

The bile metabolism is the most sensitive mechanism but offers technical difficulties in its tests. The liver is involved in the synthesis of bile salts, the formation of cholesterol in the bile and the manufacture of bile pigments. A diminution of bile salts in the gastrointestinal tract may easily cause symptoms through impairment of its functions in the emulsification of fats, the activation of lipase, the increased motility of the intestine, and the increased absorption of the fat-soluble vitamins. Pigment probably comes largely from the blood, and the site of synthesis is more probably in the reticuloendothelial system of the liver and spleen than in the bone marrow. Dr. Ravdin suggested at this point that a ten-day study of stool specimens for urobilin may be a means of differentiating carcinoma of the head of the pancreas from an obstructing stone, for in the latter this substance will be found in at least some of the samples.

The detoxifying function of the liver has been used in the hippuric acid test to measure hepatic competence, but a normal test may be found with liver destruction of 70 per cent. Among the excretory functions are those associated with the bile pigments and other dyes. The bromsulfalein test is the most sensitive test of excretion. It was concluded that a combination of all these indices offers a clue to the liver function, which may or may not be substantiated by the subsequent course. In the treatment of increased prothrombin time, it was found that approximately 70 per cent of patients responded to both the natural and synthetic vitamins.

Studies on various types of bile indicate that normally the calcium, chloride and bicarbonate contents are similar to that of serum, whereas the cholesterol is not esterified. In the gall bladder, or storehouse, base and bile salts are concentrated while the acid components are decreased. In gall-bladder disease this concentration and decrease, respectively, are not maintained, although in severe affections there may be an actual increase of fluid and an inversion of the expected ionic relations. These conditions offer a nidus for stones, for cholesterol is in equilibrium only when salts are present in proper amounts.

It was pointed out that in cholecystograms one should observe granules of opaque substance in the ascending colon to make certain that the gall bladder has had an opportunity to excrete the dye, for this is also excreted normally in the ascending colon.

As evidence that sensory stimuli from the gall bladder may simulate cardiac pain, three cases were cited of a proved inter-relation of anginal symptoms with stimuli from the gall-bladder region.

As the result of studies on the protection of the liver from toxic substances, the old concept of employing high-carbohydrate and low-fat diets has been supplanted. It was determined that the degree of hepatic injury is roughly proportional to the lipid content and that a high glycogen content offers no protection if this level is high enough. An increased protein on the other hand, although it fails to prevent liver damage, does halve the amount of necrosis. It was found during the course of these experiments that a sterile fixation abscess produced with sodium ricinoleate prevents hepatic injury to any extent with any toxin. Oral protein does not have this effect because the circulating protective agents are formed only when the toxin is administered, instead of before that time. Any sterile abscess will have the same effect, the main problem being to rid the liver of fat at the expense of protein. In regard to the proper preoperative and postoperative management of biliary cases, it was pointed out that forced feeding, with emphasis on carbohydrates and vitamins, is the best regimen for preserving glucose and diminishing lipids. Intravenous glucose is insufficient even for caloric requirements, and serves only temporarily to spare the protein stores of the body. The reappearance of the synthesis of bile salts following a common-duct operation may take many days, and bile feeding should be carried on during this entire period. If these salts remain low for more than twenty days, the patient will undoubtedly remain dyspeptic, owing to poor biliary function. It has been shown that the best protein for liver regeneration and protection is casein, whereas gelatin is the worst.

ALPHA OMEGA ALPHA

At a regular meeting of the Harvard Chapter of Alpha Omega Alpha on March 13, Dr. Israel S. Ravdin gave a companion discussion on "Preoperative and Postoperative Care of Patients with Biliary Tract Disease." It was pointed out that the mortality entailed in such surgery is in large part due to ill-advised or poorly administered preoperative and postoperative care. In considering the preoperative condition of the patient, one should remember that as little as 15 per cent of good liver tissue is adequate for normal values in most liver-function tests; thus incipient incompetence may be overlooked, and a patient thrown into insufficiency by the relatively minor trauma of the operation and anesthesia. Moderate or even severe cardiac disease, especially if demonstrable only by the electrocardiograph, should not be considered a contraindication to biliary-tract surgery, if the local indications are clear, for it has been amply demonstrated that such operations may be followed by disappearance of certain pathological electrocardiograph findings.

The incidence of diabetes mellitus in chronic gall-bladder patients is double that of the general population and may lead to easy hypoglycemic shock in such people under insulin treatment, for their liver-glycogen store — a measure of function — is invariably lowered.

The hemorrhagic diathesis of obstructive jaundice is now known to be associated with a decreased prothrombin level, which is conditioned by poor absorption of vitamin K from the intestinal tract in the absence of bile salts. Certain patients — probably those with severe liver injury — are unresponsive to any form of vitamin K therapy, and this number of complete failures is about 18 per cent. 2-methyl-1, 4-naphthoquinone has been proved one hundred to one thousand times more potent in restoring the prothrombin time to normal than the naturally occurring vitamin K. The prevention of a hemorrhagic tendency lies in frequent prothrombin-time determinations, the administration of vitamin K substrates with bile salts preoperatively and for ten to fourteen days postoperatively and transfusions of blood high in prothrombin value, when other measures fail. It was emphasized that hemorrhages may still occur after jaundice has disappeared.

Another factor that may materially alter the mortality

is the decrease of liver damage by the choice of suitable anesthetics and the preoperative liver build-up. Probably any inhalation anesthesia causes some injury, and anoxia and chlorine derivates produce the most damage. Therefore, the condition of the liver at the time of operation is the most important conditioning factor. It has been amply demonstrated that a liver high in available protein and low in lipid content affords the maximum protection against liver necrosis, if not damage. The deposition of liver glycogen is advantageous only if it displaces fat and protects the liver stores. Preoperative starvation tends to cause a high-lipid, low-protein content, and no amount of glycogen can protect a liver in which the fat content is above normal. Parenteral glucose alone cannot maintain adequate hepatic stores, and one should strive to force a high-protein, high-carbohydrate diet as soon as possible postoperatively. A diet consisting of 70 to 75 per cent carbohydrate, 20 to 25 per cent protein and less than 5 per cent fat offers the best conditions to combat damage and favor repair of the liver. The diets should be individualized for taste. Cottage cheese, with its high casein value, has been found an excellent food, and bananas have also been extensively used. Patients are given a 2600 to 4000 calorie diet a day and are actually forced to eat. Re-enforcement with plasma and intravenous fluids is helpful and sometimes necessary when patients are unable to retain food administered orally.

The only advice issued concerning the operative technic was to reveal accurately the vascular as well as the ductal systems, so that injury to or ligation of an anomalous hepatic artery, a procedure that inevitably results in death from liver insufficiency in experimental animals could be obviated.

Postoperatively, it is essential not to allow bile to be lost to the exterior in view of its important extrahepatic functions. Parenteral fluids cannot replace this loss. An attempt should be made to force it back into the duodenum by keeping the catch bottle at body level immediately after the operation, and then, by raising the bottle later, gradually to overcome the tonus. If this cannot be accomplished, bile should be fed. This can be done by collecting the bile when it begins to appear normal and then lyophilizing it.

The second important postoperative procedure is the early feeding of proper diets as described above. During regeneration, nucleoprotein is most important, and consequently the casein should be supplemented with large amounts of purines, especially liver. Finally, cholangiograms should be performed early so that immediate reoperation may be carried out in the event that a common-duct stone may be removed early, rather than caring for a stricture later. There may be enough residual liver damage in cases of long-standing disease so that there is temporary or even permanent decrease of gastrointestinal function as a result of decreased bile salts. When this diminution occurs, indigestion will remain after the gallbladder colic has disappeared. In conclusion, Dr. Ravdin cited his mortality of 1.5 per cent in 604 cases for a ten-year period of all cases that were nonmalignant or not previously operated strictures. The decrease in mortality of comparable cases since the introduction of these newer preoperative and postoperative concepts was the most striking feature.

NOTICES

ANNOUNCEMENT

Dr. Henry L. Cabitt announces the removal of his office from 475 Commonwealth Avenue, Boston, to 520 Beacon Street, Boston.

EXAMINATIONS FOR APPOINTMENTS IN THE MEDICAL CORPS OF THE UNITED STATES NAVY

Examinations for appointments as assistant surgeon (lieutenant, junior grade), United States Navy Medical Corps, will be held at all the large naval hospitals and the Navy Medical Center, Washington, D. C., on August 11 to 15, inclusive, October 6 to 9, inclusive, and January 5 to 9, 1942, inclusive.

Applicants for appointment as assistant surgeon must be citizens of the United States, more than twenty-one but less than thirty-two years of age at the time of acceptance of appointment, and graduates of Class A medical schools who have completed at least one year of intern training in a hospital approved by the Council on Medical Education and Hospitals of the American Medical Association.

Examinations for appointment as acting assistant surgeon for intern training in naval hospitals accredited for intern training by the Council on Medical Education and Hospitals of the American Medical Association will also be held on June 23 to 26, inclusive, October 6 to 9, inclusive, and January 5 to 9, 1942, inclusive. Applicants for appointment as acting assistant surgeon for intern training must be citizens of the United States, more than twenty-one but less than thirty-two years of age, and members of the junior or senior classes in Class A medical schools. After twelve months of intern training, acting assistant surgeons may apply for appointment as assistant surgeons in the United States Navy.

A circular of information regarding these examinations, physical requirements, rates of pay and promotion and retirement data may be obtained from the Bureau of Medicine and Surgery, Navy Department, Washington, D. C., on request. Applications for authorization to take the examination must be received at the Bureau of Medicine and Surgery three weeks before the examination.

Medical officers of the United States Navy are encouraged to develop a specialty, and are assigned, if their interest in the specialty warrants such action, to postgraduate instruction in the large naval hospitals, the Navy Medical Center, Washington, D. C., and civilian medical centers. Some of the specialties in which medical officers may seek qualifications are: surgery, medicine, ophthalmology, otolaryngology, roentgenology, laboratory, pathology, public health, psychiatry, deep-sea diving, aviation medicine (flight surgery), gas warfare, tropical medicine, medical research and so forth.

SOCIETY MEETINGS AND CONFERENCES

Calendar of Boston District for the Week Beginning Sunday, June 15

Monday, June 16

12:15–1:15 p.m. Clinicopathological conference. Peter Bent Brigham Hospital amphitheater.

Tuesday, June 17

12:15–1:15 p.m. Clinicoroentgenologic conference. Peter Bent Brigham Hospital amphitheater.

Wednesday, June 18

•12 m. Clinicopathological conference. Children's Hospital.

•Open to the medical profession.

June 16–July 25 — Massachusetts Institute of Technology. Page 996, issue of June 5.

June 22–24 — Maine Medical Association. Marshall House, York Harbor, Maine.

September 1–5 — American Congress of Physical Therapy. Page 997, issue of June 5.

SEPTEMBER 1–5 — American Occupational Therapy Association. The Mayflower, Washington, D. C.

OCTOBER 13–24 — 1941 Graduate Fortnight of the New York Academy of Medicine. Page 834, issue of May 8.

OCTOBER 14–17 — American Public Health Association. Page 579, issue of March 27.

APRIL 20–24, 1942 — American College of Physicians. Page 996, issue of June 5.

BOOK REVIEWS

Controlled Fertility: An evaluation of clinic service. By Regine K. Stix, M.D., and Frank W. Notestein, Ph.D. 8°, cloth, 201 pp., with 56 tables and 16 illustrations. Baltimore: William and Wilkins Company, 1940. $3.00.

This is the most important, original study of clinical contraception ever made in this or any other language. Dr. Stix, a physician on the research staff of the Milbank Memorial Fund, started to follow up a thousand women who had visited the Sanger Bureau from the Bronx in 1931 and the first half of 1932. She located 991 patients, interviewed them personally and secured a detailed and complete history. After a description of the group studied and a consideration of the representativeness and accuracy of the data, the authors launched at once into the reproductive experience of the group prior to clinic attendance. It has long been suspected by competent students of contraception that the early estimates by some clinic physicians of the reliability of contraceptives in preclinical use were worthless. Here it is shown that such "untutored" or "folk" practices are extraordinarily reliable, varying in effectiveness from 86 (condoms) to 61 (douches) per cent. It is interesting that after patients abandoned the clinic method for one more acceptable to their individual needs, they practiced their habitual forms more effectively than prior to the clinic visit.

More than 40 per cent of the couples started practicing contraception immediately after marriage. After the first pregnancy the proportion doubled, and continued to increase as the duration of marriage lengthened. Couples more recently married took up contraception sooner than older couples. White-collar workers used "folk" contraceptives much more effectively than laborers. Had the average couple not restricted family size in the nine years of married life preceding the first clinic contact, the wife would have had five pregnancies. Since almost all couples used some method relatively early, the effectiveness was such that the average number of preclinical pregnancies per woman was only three. Only 70 per cent of the pregnancies resulted in live births; 20 per cent of the women were illegally aborted.

It has been demonstrated several times before that, as pregnancies increase, the risk of induced abortion is greater. Here there is confirmation of the fact. The rate for first pregnancies was 5 per cent; for pregnancies after the fifth, 50 per cent. Almost 40 per cent of the patients had accidental pregnancies in the two years immediately preceding their initial clinical contact. This was one of the circumstances that brought them to the clinic. They not only wanted a more reliable method, but one that would permit more satisfactory coital relations.

Most of the women who discontinued the clinic prescription did so within a month. At the end of that time only 60 per cent were still using the clinic's prescription exclusively and regularly. Another 15 per cent were using it in part or alternating it with a habitual method. By fifteen months slightly over a third were using the clinic's method exclusively, and less than one half were using any part of it.

The high effectiveness of the clinic prescription is demonstrated by the fact that there were only 9 pregnancies in each 100 years of exposure for the whole group. But it is a new finding of this book that the rates of effectiveness of variations of the clinic method "did not differ significantly from those for the continuous and exclusive use of the prescribed contraceptives. . . ." Pregnancy rates with each kind of contraceptive after the patients attended the clinic were only half as high as the rates for the same devices prior to clinical attendance.

There are many practical tips in this book for the busy practitioner who has to deal with contraception. One is that it is much more important to induce a patient to adopt a method that will be continuously used than to urge the vaginal diaphragm and jelly on those who prefer another method. In other words, the absolute effectiveness of the method is less important than its acceptability to the patient.

The chapter, "Birth Control and Population Trends," is brilliant, and, on the whole, sound. There might be differences of opinion among experts with reference to the interpretation of certain details, but the broad outlines of the position there taken are certainly well founded.

The fifty-six tables and sixteen illustrations in this book assist materially in telling its story.

The Extra-Ocular Muscles: A clinical study of normal and abnormal ocular motility. By Luther C. Peter, M.D., Sc.D., LL.D. Third edition, thoroughly revised. 8°, cloth, 368 pp., with 147 illustrations and 5 color plates. Philadelphia: Lea and Febiger, 1941. $4.50.

The third edition of *The Extra-Ocular Muscles* is arranged in six parts, dealing respectively with anatomy and physiology, heterophoria, concomitant squint, paralytic squint, nystagmus and surgical technic. Important additions have been made to Parts III, IV, V and VI.

In Part III, which deals with concomitant squint, the author stresses the importance of the earliest application of measures to combat amblyopia, to correct anisometropia and aniseikonia, and to assist by orthoptic training in the development of the fusion faculty, and he reviews the indications for surgery on the eye muscles.

In Part IV, dealing with paralytic squint, a valuable section is devoted to a discussion of palsies of supranuclear origin.

Part V, written by the author in collaboration with Dr. Joseph C. Yaskin, professor of neurology at the Graduate School of the University of Pennsylvania, analyzes clearly the ocular, vestibular, and neurologic origins of nystagmus, and suggests the value of using the ocular micrometer in the corneal microscope and slit lamp for the careful study of the character and amplitude of nystagmus.

In Part VI, dealing with surgical technic, the author frankly appraises the value of surgical procedures, and adds his comments about ptosis operations, and his method of resection of ocular muscles to expedite surgery for retinal detachment and for exploration of the orbit in cases of neoplasm.

Blood Pressure Study. 1939. 4°, cloth, 69 pp. New York: compiled and published by The Actuarial Society of America and The Association of Life Insurance Medical Directors, 1940. $4.00.

This volume consists almost entirely of mortality tables based on nearly 50,000 deaths corresponding to about 1,309,000 life-insurance policies. Of greatest interest are those showing the ratio of actual to expected deaths from various causes. Tables G to M show that the higher

a man's blood pressure at the time he takes out life insurance the greater are his chances of subsequent death at any given age from cardiovascular renal disease, cirrhosis of the liver, cancer, pneumonia or tuberculosis. The likelihood of his committing suicide, however, is progressively less the higher his blood pressure when he paid his first premium (Table N). Does this mean anything, and if so, what?

Properly understood and evaluated, life-insurance mortality studies are of very great value. Those who compile them would be quick to agree, however, that the process of drawing clinical conclusions from them is full of pitfalls for the unwary. Long ago, a comparison of European insurance mortality figures with those collected by American companies indicated that American men lived much longer than European men. Although this may have been due in part to the salubrity of our climate and form of government, Emory McClintock pointed out that one potent cause of the great difference was that, whereas European offices generally awaited applications, which are commonly prompted by some sense of need for insurance, the custom of American companies was (and is) "actively to solicit business through agents." On the average, he said, men who are induced only by persuasion to insure their lives are better risks than those who voluntarily apply.

There are many other factors to be considered in the evaluation of insurance mortality figures besides the effect of selection of risks. In the present connection, for example, one might raise the question of the validity of causes of death derived from death certificates, or the extent to which the transient blood-pressure level may be treated as a statistical character.

Nevertheless, much valuable information is contained in the archives of insurance companies, and all analyses of the available material are welcome.

Treatment in General Practice: Surgery. Articles republished from the *British Medical Journal.* Vol. IV. 8°, cloth, 562 pp., with 143 illustrations. London: H. K. Lewis and Company, Limited, 1940. 16s net.

The *British Medical Journal* has published a number of articles on treatment, part of which are incorporated in this fourth volume of a series. It deals with surgery in general practice and reviews the latest treatment in some of the commoner surgical affections. A large section of this book is devoted to fractures and traumatic surgery. There are other chapters on such varied subjects as pruritus ani and prostatectomy.

The principal features of this book are a simplicity and clarity of presentation and a minimum of detail. It is designed to instruct the busy practitioner who can read only a chapter or a paragraph between patients. The material offers nothing startling or new in therapy, but is more in the nature of a review of accepted practice.

For the American practitioner, some of the specific remedies will be difficult to understand, because of the proprietary names used in their designation.

Diagnosis and Treatment of Arthritis and Allied Disorders. By H. M. Margolis, M.D. 8°, cloth, 551 pp., with 140 illustrations. New York: Paul B. Hoeber, Incorporated, 1940. $7.50.

This volume, written primarily for the family physician, attempts to present, in a concise manner, present-day knowledge of rheumatic diseases. The author has accomplished this as well as had been done in any volume on arthritis yet published. The arrangement is logical, and the pictures are good and well chosen. For those who wish a fuller discussion, there is an adequate list of references. The book is well written.

It is unfortunate, however, that the men who write about arthritis consider it necessary to discuss all the related conditions that appear in the joints. In this book, two fifths of the pages are taken up with a discussion of orthopedic conditions that should have been left to treatises on orthopedic surgery. The book should be a helpful one for the busy family physician who is called on particularly for the early treatment of patients suffering from rheumatic diseases.

The Endocrine Glands. By Max A. Goldzieher, M.D. 8°, cloth, 916 pp., with 271 illustrations. New York: D. Appleton-Century Company, Incorporated, 1939. $10.00.

For one person to write a textbook of endocrinology is to attempt what is almost impossible; the author has made this attempt, but rather unsuccessfully, according to the opinion of the reviewer. Side by side with much material that has been handed down from one textbook to another appear quite up-to-date conceptions of the pathogenesis of certain disorders; it is too bad that many concepts which have not been scientifically substantiated through the passage of years have been allowed to remain. Very often cases referred to in the text are found on careful study not to illustrate the citation. With all these criticisms, it must be said that the prefacing paragraphs on embryology and physiology of the gland under discussion are excellent; in the bibliography, there seems to be an excellent introduction to the German literature, with which the author is undoubtedly very familiar.

Vitamin Therapy in General Practice. By Edgar S. Gordon, M.D., and Elmer L. Sevringhaus, M.D. 8°, cloth, 258 pp., with 35 illustrations. Chicago: Year Book Publishers, Incorporated, 1940. $3.25.

This book presents to the clinician in a concise manner the information he needs to understand nutritional requirements and deficiencies. The vitamins are presented as a specific chemical compound playing particular roles in cellular physiology. The reader will realize that vitamins no longer are mysterious, unknown substances that can be measured only in terms of some arbitrary unit related to their biologic activity in some convenient experimental animal. As known chemical compounds, their requirements and therapeutic doses may now be designated simply in terms of milligrams. The clinician should be the first to welcome and utilize this simplification, which has been made possible by advancing knowledge.

The chapters on minerals, proteins, carbohydrates and fats present much that will refresh and supplement the clinician's knowledge. The discussion concerning low-carbohydrate versus high-carbohydrate diets in diabetes mellitus is both sound and needed. The chapter on weight control places the desired emphasis on dietary factors and control at a time when endocrine factors and therapy are so often stressed.

One regrets that the high standard of up-to-date knowledge set throughout most of the book is injured by such statements as: that a plasma ascorbic acid concentration of "from 1.0 to 0.5 mg. per cent may be considered as borderline and below 0.5 mg. a definite deficiency may be assumed to exist"; or that calcium lactate and gluconate are better sources of bone and tooth salts than calcium phosphate. And one regrets also that the high standard of accurate information is marred by an unsubstantiated

claim concerning the efficacy of vitamin P in purpura and by the statements that the characteristic laboratory finding during active rickets is primarily a fall in "blood calcium level" and that the phosphorus "in the blood" also falls, but usually does not go below 2.5 to 3.0 mg. per cent.

In spite of these rare flaws, the reader will both enjoy and profit by reading this book. And, if in reading the last chapter he is shaken from his complacent belief that this nation is well fed, his reading may be of much benefit to many.

A Pioneer Doctor in Old Japan: The story of John C. Berry, M.D. By Katherine Fiske Berry. 8°, cloth, 247 pp., with 20 illustrations. New York: Fleming H. Revell Company, 1940. $2.50.

Dr. Berry was born in Maine, on the Kennebec River, in 1847. He studied medicine at Bowdoin College, and took an eighteen-month internship at the United States Marine Hospital in Portland. He then entered the Jefferson Medical College in Philadelphia, where he graduated with honor in 1871. After a few months of practice, he went to Japan with a mission for the American Board of Foreign Missions of Boston. He crossed the Pacific in a side-wheeler, with the girl he had recently married, and started to work in Kobe, as a successor to another American physician, Dr. James C. Hepburn, who had written the first Japanese-English dictionary.

This book tells of Dr. Berry's long service in Japan. The latter part of his life was spent in Worcester, where he practiced as an otolaryngologist, until his eighty-eighth year. This charming biography is written by his daughter. In it will be found an exceptional picture of Japan and the splendid medical work done by missionaries such as Dr. Berry. Japan will not soon forget the life of this extraordinary person, and Americans may well be proud of a compatriot who did so much for a foreign country.

History of Pharmacy: A guide and a survey. By Edward Kremers, Ph.G., Ph.M., Ph.D., Sc.D.; and George Urdang, Ph.G., D.Sc. Nat. 8°, cloth, 466 pp., with 30 illustrations. Philadelphia: J. B. Lippincott Company, 1940. $4.50.

The work is divided into three parts, the third of which, having to do with pharmacy in the United States, is the first comprehensive treatment of that particular period in pharmacy. The first two parts, comprising 108 pages, deal with the early history of pharmacy and the rise of professional pharmacy in the various countries of Europe.

The book is written from the viewpoint of teaching, and for this reason the work has been kept within a comparatively small number of pages. The various supplements add to the value of the book, and the bibliography of thirty pages documents the authors' statements.

Modern Drug Encyclopedia and Therapeutic Guide. By Jacob Gutman, M.D., Phar.D. Second edition. 8°, cloth, 1644 pp. New York: New Modern Drugs, 1941. $7.00.

This encyclopedia is arranged on the same plan as the *Pharmacopoeia of the United States.* It presents the descriptions of 11,114 modern, ethical medicinal preparations in 15,629 forms, all of which are not in the pharmacopoeia. It has two comprehensive indices, one of drugs and general matters, and the other a therapeutic index.

This book should be valuable to those concerned with proprietary drugs and medicinal preparations.

Progress in Medicine: A critical review of the last hundred years. By Iago Galdston, M.D. With a foreword by Henry E. Sigerist, M.D. 8°, cloth, 361 pp. New York: Alfred A. Knopf, 1940. $3.00.

The author offers to students of science and the history of medicine an instructive and challenging account of the last hundred years of progress in medicine. It should be noted that he not only presents conclusions that have crystallized out of the controversies of other times, but also gives careful consideration to living issues. After carefully reading this book, students will come to see that many facts that the labors of earlier generations brought to light were at best provisional, and that this historical lesson is vitally applicable in modern times. The discussion is presented in clear, terse style and with a scholarly regard to historical perspective. A chapter, "The Genius of Pasteur," is preceded by "The Preludes to Pasteur" and followed by "The Pasteurians." Then follow chapters entitled "The Reign of the Microbes," "The Challenge of Nutrition," "The Inner Realm and the Endocrines," "From Mesmer to Freud," "Freud and Modern Psychiatry," "A Century of Clinical Progress" and "Whither Medicine?" This adroitly handled account of medicine during the last hundred years is strongly recommended for careful study to undergraduates and to graduates in science.

Complete Guide for the Deafened. By A. F. Niemoeller, A.B., M.A., B.S. With a foreword by Harold Hays, M.D. 8°, cloth, 156 pp. New York: Harvest House, 1940. $3.00.

This book is well written and in a language that the lay person can understand. It describes the causes of deafness and the relation of various diseases to deafness. It also discusses the effects of explosive sounds and operations of the ear on hearing. There are good sections on hearing tests, the use of the audiometer and the methods of retraining the deaf. The book should be of great value to the otologist, since it gives the viewpoint of the deafened person and also outlines the proper psychologic approach to be used with the deaf person.

Foreign Bodies Left in the Abdomen. The surgical problems: cases, treatment, prevention. The legal problems: cases, decisions, responsibilities. By Harry Sturgeon Crossen, M.D., and David Frederic Crossen, LL.B. 4°, cloth, 762 pp., with 212 illustrations, including 4 color plates. St. Louis: The C. V. Mosby Company, 1940. $10.00.

The authors have put a great deal of painstaking effort into the writing of this book. It is a medicolegal exposition on the problems of foreign bodies left in the body at the time of operation, and is written by a surgeon and a lawyer. It is more in the nature of an encyclopedia and reference book for the use of a person with such a problem in hand. The great detail of the case reports and of the individual experiences precludes a cover-to-cover reading simply for interest in the material presented.

The most valuable part of the volume to the practicing surgeon is that which considers prevention of these unfortunate accidents. Use of the continuous-strip instead of individual sponges is advocated as the safest method for packing away the abdominal contents. A chapter on ingested foreign bodies is interesting because it again calls attention to the amazing array of objects that the alimentary canal can expel spontaneously.

This book will find its greatest usefulness in the reference library for those who are called on to assist in the legal solution of these cases.

A Surgeon Explains to the Layman. By M. Benmosché, M.D. 8°, cloth, 317 pp., with 62 illustrations. New York: Simon and Schuster, 1940. $3.00.

In this book the author has attempted to popularize the commoner surgical procedures. To the reviewer, it seems that the type of person who will read such a volume for self-education is the one who is intelligent enough to consult his physician for unexplained symptoms. In such cases, the physician can describe a specific disease for the patient better than any book. Other classes of patients are better reached through magazines, cinema and radio than through such a medium as this. They are the ones who will at best understand and follow such a simple rule as, "Don't take a laxative for pain in the abdomen; see a doctor first."

The reviewer believes that the author has made an effort to popularize a subject that does not lend itself well to such a purpose.

The 1940 Year Book for Industrial and Orthopedic Surgery. Edited by Charles F. Painter, M.D. 12°, cloth, 484 pp., with 299 illustrations. Chicago: The Year Book Publishers, Incorporated. 1940. $3.00.

In this small book, the author has attempted to condense the worthwhile contributions in orthopedic surgery of the past year, and he has succeeded as well as possible with so large a task. The book is clearly and concisely written. The arrangement is commendable, and the subject matter is well outlined and indexed.

Unfortunately, what constitutes a contribution to orthopedic surgery and what amount of emphasis each topic should be given are matters on which each orthopedic surgeon differs. Tuberculosis receives more attention than its present-day importance seems to merit, and congenital and developmental deformities are given very brief notice. The section on fractures is one of the best in the book. The same thing might be said of the final chapter on industrial medicine.

The style is good, a rare thing in medical treatises of this generation. The book is interesting reading. It is an excellent compendium of present-day orthopedic practice and should prove helpful to all orthopedic and industrial surgeons.

Psychotherapy: Treatment that attempts to improve the condition of a human being by means of influences that are brought to bear upon his mind. By Lewellys F. Barker, M.D. 12°, cloth, 218 pp. New York: D. Appleton-Century Company, Incorporated, 1940. $2.00.

Few men in America are so well qualified by virtue of wide medical experience to discuss psychotherapy as Dr. Barker. This book is ample proof that he has dealt with patients in various stages of mental disturbance for a long and profitable period.

Dr. Barker points out, soon after defining his understanding of the title, the importance of making comprehensive diagnostic studies on which adequate treatment can be based. He believes that a knowledge of the heredity and environment of a patient is of extreme importance in mapping a plan of specific treatment. The methods of mental therapy, such as suggestion and hypnotism, persuasion, rest and isolation, are capably reviewed before he discusses the more comprehensive treatments associated with the name of a single psychiatrist. Psychoanalysis is considered not only as a psychologic entity and technic but also as a historical landmark. The dependence of the "psychological analysis" of Janet, the "individual psychology" of Adler and the "analytical psychology" of Jung on the basic technic is extensively elaborated. The contributions of Stekel and Rank are reviewed.

Psychotherapy in the various ages of life (childhood, adolescence, adulthood, in later life and old age) forms a large part of the volume, but psychotherapy as it relates to the treatment of organic and functional disorders is not neglected. A chapter on the future of psychotherapy is inspiring and should be read by those physicians who believe that psychologic treatment has reached a cul-de-sac beyond which no further application can be envisioned.

This book warrants the attention of physicians who do not specialize in psychiatry, and of medical students, social workers and persons who wish to obtain a broad view of the field or who wish a starting point for more intensive studies.

Fractures and Dislocations for Practitioners. By Edwin O. Geckeler, M.D. Second edition. 8°, cloth, 314 pp., with 267 illustrations. Baltimore: Williams and Wilkins Company, 1940. $4.00.

The first edition of this book, published in 1937, had of necessity to be soon followed by a second, because so rapid a pace is being set by those who are interested in the treatment of injuries to bones. It is clearly written, well illustrated and full of practical suggestions concerning methods of treatment. The emphasis laid on aftercare should be taken to heart, for more depends on it than some recognize. The modern operative approach to such cases as are better handled by open methods is well defined, and the technic is outlined.

This volume can be enthusiastically recommended to all who wish a concise statement of the essentials of the treatment of fractures and dislocations.

Taber's Cyclopedic Medical Dictionary. By Clarence Wilbur Taber. 12°, cloth, 1372 pp., with 273 illustrations. Philadelphia: F. A. Davis Company, 1940. $2.50.

This small dictionary of 50,000 words is a new publication in the field of dictionaries and is not a revision of the author's digest of medical terms, which was published in 1937.

Of particular interest are the appendices, consisting of a glossary of Latin medical words, definitions of prefixes and suffixes and "the interpreter," which is a phrase book written in five languages and especially arranged for diagnosis.

Dizionario di medicina per medici e famiglie. By Dott. Giulio Casalini. F°, cloth. Vol. 1, 868 pp., with 1550 illustrations and 24 color plates; Vol. 2, 917 pp., with 1912 illustrations and 35 color plates. Torino, Italy: Corso Taffaello, 1939. Lire 300.

This well-illustrated dictionary is encyclopedic in character. There are 8000 subjects, ranging in size from a few lines to a number of pages, and illustrated with many figures and plates, some of which are in color.

The work is well printed on good paper and reflects the current aspect of Italian medicine. The dictionary was written for both physicians and the laity, and Dr. Casalini was assisted by a corps of twelve eminent Italian professors and doctors.